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ASSOCIATION BETWEEN BIRTH DEFECTS AND EXPOSURE TO AMBIENT VINYL CHLORIDE

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16. ABSTRACT

To better define the association between exposure to vinyl chloride monomer (VCM) and the occurrence of birth defects, this epidemiological study was made in Shawinigan, Quebec, Canada, where a vinyl chloride polymerization plant has operated since 1943. Birth-defect rates in Shawinigan during the last 15 years were compared with rates in three other communities, and seasonal and spatial variations in Shawinigan's birth-defect rate were correlated with estimated VCM concentrations in the environment.

Shawinigan had an excess of birth defects which fluctuated seasonally in a way that could correspond to changes in VCM concentration in the environment. Mothers who gave birth to malformed children were younger on average in Shawinigan than in the comparison communities. However, there was no excess of stillbirths in Shawinigan, the excess of birth defects involved most systems, and variation in birth-defect rates among school districts could not be accounted for by estimates of VCM in the atmosphere.

The occupational and residential histories of parents who gave birth to malformed infants were compared with those of parents of normal infants. The two groups did not differ in occupational exposure or closeness of residence to the vinyl chloride polymerization plant.

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FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the public health and welfare and the productive capacity of our Nation's population.

The Health Effects Research Laboratory, Research Triangle Park, conducts a coordinated environmental health research program in toxicology, epidemiology, and clinical studies using human volunteer subjects. These studies address problems in air pollution, non-ionizing radiation, environmental carcinogenesis and the toxicology of pesticides as well as other chemical pollutants. The Laboratory participates in the development and revision of air quality criteria documents on pollutants for which national ambient air quality standards exist or are proposed, provides the data for registration of new pesticides or proposed suspension of those already in use, conducts research on hazardous and toxic materials, and is primarily responsible for providing the health basis for non-ionizing radiation standards. Direct support to the regulatory function of the Agency is provided in the form of expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of health care and surveillance of persons having suffered imminent and substantial endangerment of their health.

Vinyl chloride is known to be carcinogenic for humans and other animals. Unlike many carcinogens, it produces a highly specific effect: occupational exposure results in a high incidence of angiosarcoma of the liver. Thus, possible effects on the health of the general population (such as cancer or birth defects) of low-level exposure to ambient vinyl chloride are of concern.

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ABSTRACT

The objective of this research program was to better define the association between exposure to vinyl chloride monomer (VCM) and the occurrence of birth defects in communities near VCM-emitting plants.

This epidemiological study took place in Shawinigan, Quebec, Canada, where a vinyl chloride polymerization plant has operated since 1943. Among workers at this plant, ten cases of angiosarcoma of the liver have been reported. The study assessed the birth-defect rates in Shawinigan during the last 15 years, compared these rates with those of three other communities, and correlated seasonal and spatial variations in birth-defect rates with estimated VCM concentrations in the environment. (VCM concentrations were estimated in two concomittant environmental studies.) Also, the occupational and residential histories of parents who gave birth to malformed infants were compared with those of parents of normal infants.

Shawinigan was found to have an excess of birth defects. The excess fluctuated seasonally in a way that could correspond to changes in VCM concentration in the environment. The excess in birth defects involved most systems, and mothers from Shawinigan who gave birth to malformed children were younger on average than mothers from the comparison communities who also gave birth to malformed infants.

Variation in birth-defect rates among school districts could not be accounted for by estimates of VCM in the atmosphere. Parents of malformed children did not differ from parents of normal children in occupational exposure or closeness of residence to the PVC plant. There was no excess of stillbirths in Shawinigan, and the mothers of malformed children did not differ in abortion rate from mothers of normal infants.

In conclusion, an association between VCM in the environment and excess birth defects in this community cannot be substantiated at present. The possibility of such an association remains, but its existence can be determined only by monitoring VCM concentrations in the air and birth defects in the community for several years.

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CONTENTS

SECTION		PAGE
Foreword		iii
Abstract		iv
Figures		vi
Tables		vi
Acknowledgments		ix
1.	Introduction	1
2.	Conclusions and Recommendations	3
3.	Methods	4
	Study population and observation periods	4
	Definition of a birth defect	4
	Comparison communities	5
	Data sources	6
	Identification of birth-defect cases	6
	Selection of control infants	6
	Data-collection forms	7
	Seasonal and spatial variation in birth-defect rates	7
	Estimates of vinyl chloride exposure	7
	Quality-control procedures	8
	Analysis	9
4.	Results	10
	Birth defects in Shawinigan	10
	Shawinigan and the comparison communities	12
	Seasonal variation in birth-defect rates	18
	Spatial variation in birth-defect rates	20
	Case-control study	25
5.	Discussion	33
	Previous studies	33
	The present study	34
References		37
Appendices		
A.	Details of methods	A-1
B.	Data	B-1
C.	Summary of a study of the feasibility of measuring ambient vinyl chloride concentrations in Shawinigan	C-1
D.	Dispersion patterns of vinyl chloride emitted by B.F. Goodrich Co., Ltd., Shawinigan, Quebec	D-1

FIGURES

NUMBER PAGE

1 Monthly distribution of birth-defect rates in Shawinigan and the comparison communities 20

2 Residences of birth-defect cases and controls at conception 32

TABLES

1 Distribution of malformed children by sex and affected system (Shawinigan) 10

2 Distribution of malformed children by year and affected system (Shawinigan) 11

3 Distribution of malformed children by affected system and mother's age at delivery (Shawinigan) 12

4 Test of difference in age of mother among affected systems (Shawinigan) 13

5 Distribution of malformed children by week of gestation and affected system (Shawinigan) 14

6 Distribution of malformed children by mother's residence at delivery, sex, and affected system 14

7 Number of malformed children observed in Shawinigan compared with expected numbers based on rates for the comparison communities 15

8 Numbers of stillbirths, with or without birth defects, and numbers expected in Shawinigan based on rates in the comparison communities 16

9 Distribution of malformed children by number of birth defects and mother's residence at delivery 16

(continued)

TABLES (continued)

NUMBER		PAGE
10	Distribution of malformed children for each year (1966-1979)	17
11	Mean age of the mother at delivery, by affected system	17
12	Mean gestational age at birth, by affected system	18
13	Distribution of malformed children by month and affected system (Shawinigan)	19
14	Distribution of malformed children by month and affected system (Drummondville and Rimouski)	19
15	Distribution of malformed children by month of birth	21
16	Estimates of parameters of seasonal variation	21
17	Distribution of malformed children by affected system and school district of mother's residence at delivery (Shawinigan)	22
18	Distributions of malformed children and birth-defect rates by school district of mother's residence at delivery (Shawinigan)	23
19	Comparison of total birth defects and CNS birth defects between school districts with high and low atmospheric vinyl chloride concentration	23
20	Comparison of total birth defects and CNS birth defects between school districts adjacent to the vinyl chloride plant and the other school districts	24
21	Comparison of total birth defects and CNS birth defects between school districts located inside and outside a one-mile radius of the vinyl chloride plant	24
22	Distribution of Shawinigan cases by type of birth defect	26
23	Distribution of cases and controls by mother's age at delivery	26
24	Distribution of cases and controls by weeks of gestation	27
25	Distribution of cases and controls by previous occurrence of birth defects in the family	27

(continued)

TABLES (continued)

NUMBER		PAGE
26	Distribution of cases and controls by number of previous pregnancies	28
27	Distribution of cases and controls by diseases of the mother during pregnancy	28
28	Distribution of cases and controls by number of previous abortions	29
29	Distribution of cases and controls by father's age at birth of malformed infant	29
30	Distribution of cases and controls by mother's occupation	30
31	Distribution of cases and controls by smoking habits of the mother	30
32	Distribution of cases and controls by father's occupation	31
33	Distribution of cases and controls by mother's residence during the first third of pregnancy	31

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SECTION 1

INTRODUCTION

Vinyl chloride monomer (VCM) is a carcinogen for both animals (1-3) and man (4-9). It is also mutagenic in man (chromosome breaks have been observed in lymphocytes of workers exposed to VCM; 10-15), in microbial tests, in Drosophila, in yeast, and in mammalian cells (16-19). VCM generates mutations primarily through one of its metabolites, chloroethylene oxide, and with the involvement of liver microsomal enzymes (17, 20-22). Although miscarriages and fetal losses among the wives of workers exposed to VCM have been studied (23), such effects remain undocumented (24, 25). VCM teratogenicity has yet to be demonstrated. VCM was present in the fetal and maternal blood and the amniotic fluid of pregnant rats after exposure to air containing VCM (26). Two studies of VCM teratogenicity in animals (26, 27) failed to demonstrate fetal malformations, but showed increased fetal mortality rates.

VCM mutagenicity and possible teratogenicity in man raised the question of the origin of birth defects in communities near VCM-emitting plants. In 1975, a high birth-defect incidence (mostly central nervous system abnormalities) was observed in three Ohio towns where VCM plants operated (28). These observations were only partly confirmed by two subsequent studies (29, 30), which failed to find any association between birth defects and either work at or proximity of residence to VCM plants. In a 1977 study comparing birth-defect rates between Shawinigan (a town in the province of Quebec with an operating VCM polymerization plant) and Drummondville (a control community), Thériault and Goulet (31) noted a higher birth-defect frequency in Shawinigan, with highest rates in the vicinity of the plant. No attempt has yet been made to associate this high frequency of birth defects with exposure to VCM.

At normal temperature and pressure, VCM is a gas; it liquifies at -13° C. It is processed into polyvinyl chloride (a plastic) through polymerization reactions in autoclaves. The gas is released into the environment during unloading, processing, and autoclave-cleaning operations. The concentration of VCM to which people in the community are exposed depends on production rate, wind direction, wind velocity, and distance from the plant.

In Shawinigan (population 27,000), a vinyl chloride polymerization plant has operated since 1943. The plant, which employs about 250 workers, currently produces 25,000 tons/yr of PVC. Ten cases of angiosarcoma of the liver have been reported among workers at this plant. In addition to the VCM plant, Shawinigan has two chemical plants, one aluminum electrolysis plant, one carbide plant, and one pulp and paper mill. A great number of pollutants are discharged into its environment. Furthermore, prevailing winds tend to blow from the several pollution sources towards the town's residential areas.

Birth defects generally show some degree of association with other risk factors such as maternal age, drug consumption, radiation exposure during pregnancy, and maternal illnesses. These factors should be considered in studies of the association of birth defects with exposure to environmental pollutants.

The purposes of this research were twofold: to confirm the existence of higher-than-expected birth-defect rates in Shawinigan and to better define the association between the occurrence of birth defects in a community and the concentration of VCM in residential areas.

This study had four specific objectives. The first was to establish birth-defect and stillbirth rates in Shawinigan and to compare these rates with those for other communities, in an attempt to confirm the high birth-defect rates previously observed in this town. The second objective was to try to correlate monthly and seasonal variations in the occurrence of birth defects with variations in vinyl chloride concentration in the environment. The third objective was to try to correlate spatial variations in birth-defect rates with estimates of VCM concentrations in the air in residential areas. The final objective was to compare a group of parents who gave birth to malformed infants with a control group of parents of normal children, with respect to several risk factors, as well as residential and occupational history.

SECTION 2

CONCLUSIONS AND RECOMMENDATIONS

The conclusions of this study can be summarized as follows:

1. For the years 1966 through 1979, there was an excess of birth defects in the population of Shawinigan, Quebec. This result is similar to the findings for four U.S. cities where vinyl polymerization plants operate.

2. Birth-defect rates underwent a seasonal variation that could correspond to variation in the concentration of VCM in ambient air.

3. Mothers from Shawinigan who gave birth to malformed infants were younger than mothers from the comparison communities who also gave birth to malformed infants.

4. The excess malformations involved not only the central nervous system, but almost all body systems.

5. No correlation was found between the spatial distribution of birth defects in the town and estimates of VCM concentrations in the air. (These estimates were based on approximate data, since information on PVC production and VCM emissions was not available.)

6. Occupational exposure as a causal agent was ruled out, because none of the parents in the study had ever worked at the VCM plant.

7. Cases and controls did not differ in distance of residence from the plant.

8. No excess of stillbirths was observed in Shawinigan, and abortion rates were no higher for mothers of malformed infants than for mothers of normal infants.

Some observations of this study support an association between VCM in the air and birth defects in the exposed community, while others tend to indicate that no such association exists. As the present results are inconclusive, the possibility that VCM generates birth defects in human communities requires further consideration. This would best be accomplished through a program during the next 5 to 10 years to monitor VCM concentration in the air and birth defects in the exposed communities.

SECTION 3

METHODS

STUDY POPULATION AND OBSERVATION PERIODS

For determining birth-defect rates in Shawinigan, the study included all stillbirths and malformed children born of mothers who resided within the city limits of Shawinigan at the time of delivery and whose delivery took place between January 1, 1966, and December 31, 1979. The case-control study was limited to a shorter period, from January 1, 1973, to December 31, 1979, because it required information based on the mothers' recollection of events during their pregnancies.

DEFINITION OF A BIRTH DEFECT

A literature search and consultation with colleagues revealed that although a congenital birth defect is something intuitively clear and simple, there is actually no agreement as to what constitutes a birth defect (see 32). For the present study, a birth defect was defined as a gross physical or anatomic developmental anomaly present at birth or detected at the hospital during the first days after delivery (within the first four to six days, on average). To be included, a birth defect had to be obvious enough not to leave doubt as to its presence. A list of the birth defects included appears in Appendix A. Congenital malformations were not categorized as "major" and "minor," as such divisions are usually arbitrary and subjective (32) and would have served no purpose in the present study.

Two main difficulties were encountered. In birth defects of the cardiovascular system, it soon became apparent that in several hospitals, no diagnoses other than murmurs of the heart were reported, whereas in other hospitals (e.g., Shawinigan), thorough investigations were conducted, leading to precise diagnoses. Heart murmurs judged severe enough were therefore included in the study, in a separate category. Severity and significance of heart murmurs were assessed by consulting the children's subsequent medical records.

Although such minor defects as metatarsus varus, calcaneum varus and valgus, metatarsus adductus, and abnormal positioning of fingers or toes were not included in the study, foot anomalies were included if they required either a cast or surgery for correction.

COMPARISON COMMUNITIES

To assess the importance of the excess of birth defects in Shawinigan, a comparison population was necessary. The provincial birth registry was not a sufficiently reliable source of information, and its data were collected in a different manner than were data for Shawinigan. Therefore, comparison communities were chosen, based on their similarity to Shawinigan in three respects: population structure, socioeconomic level, and medical-care facilities.

Since it is well established that birth defects reported from rural areas differ in type and frequency from those occurring in large cities (32), the comparison communities were selected from industrialized towns similar in size to Shawinigan. The size criteria were number of residents, annual birth rate, increment in the female population, and the shape of the age pyramid. As low socioeconomic status has been associated with high birth-defect rates (33), comparison communities also had to be similar to Shawinigan in the percentage of the labor force in various economic activities, the principal industrial activities, and the average family income. Because the availability of health professionals and medical-care facilities may influence birth-defect rates, the ratios of physicians to population and hospital beds to population were also considered in choosing the comparison communities.

Finally, environmental pollution was considered. In Shawinigan, there are three industries that emit chemicals into the environment that could potentially produce birth defects: a vinyl chloride polymerization plant, an aluminum electrolysis plant, and a chlore alkali factory. Information provided by Les Sérvices de Protection de l'Environnement du Québec and from an industrial index indicated that the chlore alkali plant in Shawinigan does not discharge organic mercury and that such discharges prior to 1966 were negligibly small. Thus, the presence of a chlore alkali plant was not used as a criterion in selecting comparison communities. The control communities were chosen to provide a control town with an aluminum plant, but no VCM-emitting plant (Baie-Comeau--Hauterive), and two control towns with neither a VCM nor an aluminum plant (Drummondville and Rimouski).

In Appendix A, Shawinigan and the comparison communities are characterized with respect to the selection criteria. Several differences remained between Shawinigan and the comparison communities: with a comparable population, Shawinigan had a lower birth rate; a larger percentage of Shawinigan's labor force was engaged in manufacturing than were those of Rimouski and Baie-Comeau--Hauterive, but a smaller percentage than in Drummondville; the average family income was lower in Shawinigan than in any of the other towns; and together with Drummondville, it had an older population. Obviously, no two towns are completely alike; we believe that these comparison communities were the best available choices.

DATA SOURCES

Information was secured from several sources. The Quebec Population Registry provided the annual numbers of births and stillbirths in the communities under study. As the 1979 data were not available, numbers were estimated from the three preceding years. The Quebec Ministry of Social Affairs provided a list of hospitals where women residing in these communities gave birth during the study period. The School Board of Shawinigan furnished the school district boundaries. Birth-defect children and controls were identified and data were collected from several medical files and hospital rosters (delivery-room daybooks, birth-defect rosters, discharge lists, mothers' and children's medical files, and birth rosters).

Most deliveries (approximately 95%) took place at one or two regional hospitals in each town in the study. The remaining deliveries took place in some 50 other hospitals. If any hospital had ten or more deliveries during the study period, it was visited and information was collected. The sixteen hospitals visited are listed in Appendix A.

IDENTIFICATION OF BIRTH-DEFECT CASES

The following procedures were used to identify infants with birth defects, collect data from hospital files, and interview designated mothers to secure additional information. Malformed newborns whose mothers resided in these communities at the time of delivery were identified from the birth-defect rosters and departure lists of the hospitals included in the study. A physician reviewed medical files to decide whether or not each infant identified met the description of a birth-defect child and should be included in the study. When there was any doubt as to the presence or severity of the malformation, the physician referred to consultation reports or specialists' advice before reaching a decision. For Baie-Comeau--Hauterive and Rimouski, the physician reviewed the files not only of known malformation cases, but of all newborns, to ensure that all cases had been reported.

For each malformed infant identified, the physician filled out Part A of the data-collection form. For the case-control study, the mothers of all affected infants and matched controls were interviewed at home by two medical students trained for this purpose, using a blind protocol. The information was recorded in Part B of the data-collection form.

SELECTION OF CONTROL INFANTS

For each confirmed case of birth defect in an infant born of a mother whose residence during the first trimester of pregnancy was Shawinigan, one control was selected from the same hospital. Each control was a live-born infant who gave no indication of congenital malformation, whose parents resided in Shawinigan, and whose birth immediately followed that of the infant with the birth defect. Certain types of malformations are reportedly more

frequent among mothers aged 40 and older, and several authors have reported sex variation in certain types of birth defect (33). Therefore, cases and controls were matched by maternal age within two years, sex of the infant, and place of residence of the mother (within the Shawinigan city limits).

DATA-COLLECTION FORMS

The forms used to collect data are shown in Appendix A. Part A was filled from hospital records and used to compare birth-defect rates between Shawinigan and the other communities. This part included items such as name and address of the parents; mother's age; child's sex and date of birth; gestational age and vital status at birth; a description of the birth defects; name of the delivering physician; number of deliveries, live births, still-births, and spontaneous abortions; and several items permitting further tracing.

Part B was used for the case-control study and was completed with the help of the mothers. It included items such as residential history of the parents, medical history of the mother before and during pregnancy, occupational history of the parents, and the mother's previous exposure to birth-defect risk factors such as viral disease during pregnancy, X-ray exposure, smoking, and alcohol and drug consumption.

SEASONAL AND SPATIAL VARIATION IN BIRTH-DEFECT RATES

For 1971 through 1977, the monthly distribution of live births was available from Government sources only for towns with populations exceeding 25,000. This excluded Baie-Comeau--Hauterive, which was considered as two separate towns with populations under 25,000. For Shawinigan, the missing information for the years 1966 through 1970 and 1978 through 1979 was secured by reviewing all birth records. Thus, monthly variation in birth defect rates was studied by comparing data for Shawinigan from 1966 through 1979 with data for Drummondville and Rimouski from 1971 through 1977.

School Districts were used as the unit of spatial distribution of birth defects. Shawinigan has 14 school districts whose boundaries are well defined. Each birth was attributed to one of these districts; numbers were estimated for the years 1975, 1977, and 1979.

ESTIMATES OF VINYL CHLORIDE EXPOSURE

Two studies were conducted to assess the vinyl chloride concentration in the air in Shawinigan. The first was a study of the feasibility of measuring VCM in the air at several locations in Shawinigan using air sampling and analysis techniques. The report of this feasibility study appears as Appendix C. Its conclusion was that environmental VCM concentrations could be

assessed by ambient air sampling at various sites in Shawinigan; depending on location, sampling detected levels up to 45 ppb. During winter, VCM tended to accumulate in the snow in the vicinity of the plant, and VCM concentrations differed between indoor and outdoor samples. The report recommended a continuous one-year sampling program with special attention to meteorologic, topographic, and demographic data to establish the true VCM concentrations to which the Shawinigan population is exposed. A more detailed summary of this feasibility study appears as Appendix C.

Considering the high cost of a comprehensive air-monitoring program and the results of this pilot study, it was decided that ambient vinyl chloride concentrations in Shawinigan should be estimated using a dispersion model based on production records and the results of previous air-monitoring activities. This study was conducted by a subcontractor; the report is included as Appendix D.

The dispersion model used the Pasquill-Gifford equations and allowed the development of isopleths of vinyl chloride concentration over Shawinigan. It employed meteorological variables (such as wind direction and velocity, rainfall, humidity, and temperature), and topographic variables (including buildings), as well as such variables as production, emissions, and plume height. The model identified an area in Shawinigan where VCM concentrations are believed to have been relatively high for a long time.

QUALITY-CONTROL PROCEDURES

The physician who identified the birth-defect cases and the medical students who interviewed the mothers were familiarized with the study. The physician had regular opportunities to discuss any problem with the physician coordinator, and the medical students were given two days of training. All information collected was reviewed by the physician coordinator, who cross-checked approximately 5% of the filled-in forms with the hospital files. Errors were detected in less than 0.5% of the reviewed material. All home interviews were checked regularly, and missing information was obtained from the respondent by telephone. On such occasions, responses to other questions were verified, revealing very few errors.

The information on the data-collection forms was coded, and the coded information was compared with the forms. The coded information was recorded on punched cards, and the printouts were checked against the coded sheets or the original forms when this was deemed necessary. No errors were subsequently detected when results were compared with the information recorded on the data-collection forms.

ANALYSIS

The following statistical tests were used in this study:

1. chi-square test (34),
2. median test (35),
3. ratio of a Poisson variable to its expectation (36),
4. relative risks and confidence interval of the relative risk (37),
5. Student's *t*-test (38),
6. test for seasonality of events (39),
7. McNemar matched-pairs test (38), and
8. Wilcoxon's signed-ranks test (34).

SECTION 4

RESULTS

BIRTH DEFECTS IN SHAWINIGAN

From January 1966 through December 1979, there were 4534 live births and 33 stillbirths in Shawinigan. Of the live infants, 150 had birth defects, and of the stillbirths, 9 had birth defects. Tables 1 through 5 summarize the birth defects found among infants born to Shawinigan residents during this period. The original data, including a list of all birth defects encountered, is given in Appendix B.

As shown in Table 1, the birth defects reported most frequently were those of the musculoskeletal system, followed by the cardiovascular, central nervous, and urogenital systems. The only statistically significant difference

TABLE 1. DISTRIBUTION OF MALFORMED CHILDREN BY SEX AND AFFECTED SYSTEM (SHAWINIGAN, 1966-1979)

	Males		Females			
Affected System	N	Rate[a]	N	Rate[a]	Total	X^2_1[b]
Central nervous	13	5.57	17	7.73	30	0.51
Cardiovascular	21	9.00	16	7.27	37	0.23
Gastrointestinal	7	3.00	4	1.82	11	0.26
Urogenital	28	12.00	2	0.91	30	19.54***
Musculoskeletal	18	7.71	31	14.09	49	3.73
Mouth and upper airways	6	2.57	11	5.00	17	1.20
Eye and ear	4	1.71	2	0.91	6	0.11
Chest	1	0.43	2	0.91	3	0.00
Syndromes and other defects	1	0.43	5	2.27	6	1.69
Total	85	36.42	74	33.64	159	0.18

[a]Number of malformed children per 1000 live births.
[b]*** indicates $p < 0.001$.

between the sexes was that boys had more urogenital malformations than did girls.

Table 2 shows the annual distribution of birth defects over the study period. The yearly numbers of malformed children per 100 births varied from 1.68 (in 1969) to 6.84 (in 1973), with an overall rate of 3.48. Detailed analysis of the monthly distribution of birth defects and the distribution by affected system for the year 1973 showed no single large increase that could account for the high rate in that year. Nor did the yearly distribution by system show any particular feature that could account for the annual variation in the overall rates. Furthermore, birth-defect rates did not consistently increase or decrease with time.

Table 3 shows the distribution of mothers' ages at delivery for each category of defect, and Table 4 compares the mothers' ages among the various types of birth defect; according to the median test, the ages did not significantly differ. The distribution of birth defects according to the

TABLE 2. DISTRIBUTION OF MALFORMED CHILDREN BY YEAR AND AFFECTED SYSTEM (SHAWINIGAN)

Year of Birth	Affected System										
	CNS	Cardio-vascular	Gastro-intestinal	Urogenital	Musculo-skeletal	Mouth & Upper Airways	Eye & Ear	Chest	Syndromes & Other Defects	Tot.	Rate[a]
1966	7	0	3	2	6	2	0	1	0	16	3.54
1967	4	3	1	3	6	2	1	0	1	13	3.52
1968	2	0	2	1	2	1	0	1	0	8	2.30
1969	1	3	0	1	0	1	0	0	0	6	1.68
1970	3	4	0	3	6	1	0	0	1	15	4.70
1971	0	2	1	1	2	0	0	1	0	6	2.08
1972	1	2	2	0	2	2	0	0	0	6	2.19
1973	2	4	1	3	4	3	1	0	1	18	6.84
1974	1	2	1	?	3	1	1	0	0	8	2.97
1975	2	3	0	4	5	2	0	0	0	15	4.40
1976	2	7	0	2	2	0	0	0	2	13	3.98
1977	2	6	0	2	5	0	0	0	0	14	4.56
1978	3	1	0	3	4	1	0	0	0	11	3.33
1979	0	0	0	3	2	1	3	0	1	10	3.11
Total	30	37	11	30	49	17	6	3	6	159	3.48[b]

[a]Number of malformed children per 100 births.
[b]$X^2_{13} = 20.75$; $p = 0.08$.

TABLE 3. DISTRIBUTION OF MALFORMED CHILDREN BY AFFECTED SYSTEM AND MOTHER'S AGE AT DELIVERY (SHAWINIGAN, 1966-1979)

Affected System	Age of Mother									
	< 20	20-24	25-29	30-34	35-39	> 40	Unkn.	Tot.	Mean	S.D.
CNS	1	17	9	3	0	0	0	30	24.63	3.44
Cardio-vascular	1	16	11	6	3	0	0	37	26.16	4.84
Gastro-intestinal	0	4	3	3	1	0	0	11	27.64	5.33
Urogenital	1	15	10	4	0	0	0	30	24.90	4.01
Musculo-skeletal	3	20	15	8	2	1	0	49	26.18	5.16
Mouth & upper airways	2	8	6	0	0	0	1	17	22.63	3.12
Eye & ear	0	5	1	0	0	0	0	6	23.83	2.93
Chest	0	3	0	0	0	0	0	3	22.33	1.15
Syndromes & other defects	0	1	3	1	1	0	0	6	27.50	4.51
Total	8	73	50	20	6	1	1	159	25.41	4.52

number of weeks of gestation at birth is shown in Table 5. Twelve of the 159 cases had less than 35 weeks of gestation; 7 of these 12 presented central nervous system (CNS) defects.

SHAWINIGAN AND THE COMPARISON COMMUNITIES

Table 6 gives the number of birth defects for each affected system according to the mother's place of residence at delivery and the child's sex. As can be seen in Table 7, the observed number of malformed children in Shawinigan was higher than would be expected based on the rate for each comparison community or for all three comparison communities together. The excess birth defects in Shawinigan occurred in the central nervous, cardio-vascular, urogenital, and musculoskeletal systems, the eye and ear, and the chest. As types of defects found in excess in Shawinigan were also the most common types of defects overall, it can be concluded that birth defects in general were in excess in Shawinigan.

The number of stillbirths did not differ between Shawinigan and the comparison communities, except for Drummondville, where the number of stillbirths was significantly higher (Table 8). The proportion of malformed

TABLE 4. TEST OF DIFFERENCE IN AGE OF MOTHER AMONG AFFECTED SYSTEMS (SHAWINIGAN, 1966-1979)[a]

Affected System	Mean Age of Mother	Standard Deviation	Number of Mothers of Age < 24.4	Number of Mothers of Age > 24.4	Age Unknown	Total
Central nervous	23.95	2.68	13	6	0	19
Cardiovascular	26.23	4.67	13	18	0	31
Gastrointestinal	29.00	5.15	1	4	0	5
Urogenital	24.67	3.89	13	11	0	24
Musculoskeletal	26.10	5.42	19	20	0	39
Mouth and upper airways	21.56	2.92	7	2	1	10
Eye and ear	25.00	2.71	3	1	0	4
Chest	23.00	0.00	2	0	0	2
Syndromes and other defects	26.00	1.00	0	3	0	3
More than one system affected	26.05	4.65	10	12	0	22
Total	25.41	4.54	81	77	1	159

[a]Median test: median = 24.4; $X^2_{13} = 12.05$; $p = 0.21$.

TABLE 5. DISTRIBUTION OF MALFORMED CHILDREN BY WEEKS OF GESTATION AND AFFECTED SYSTEM (SHAWINIGAN, 1966-1979)

Affected System	Weeks of Gestation							
	20-29	30-34	35-36	37-38	39-40	41-42	Unknown	Total
Central nervous	3	4	3	8	9	3	0	30
Cardiovascular	0	0	3	9	21	4	0	37
Gastrointestinal	0	0	2	3	5	1	0	11
Urogenital	2	1	2	8	13	4	0	30
Musculoskeletal	4	2	3	7	27	6	0	49
Mouth and upper airways	1	0	4	2	8	1	1	17
Eye and ear	0	0	1	1	3	1	0	6
Chest	0	0	0	1	2	0	0	3
Syndromes and other defects	0	1	1	2	1	1	0	6
Total	6	6	10	33	84	19	1	159

TABLE 6. DISTRIBUTION OF MALFORMED CHILDREN BY MOTHER'S RESIDENCE AT DELIVERY, SEX, AND AFFFECTED SYSTEM (1966-1979)

Affected System	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
	M	F	T	M	F	T	M	F	T	M	F	T
Central nervous	13	17	30	12	25	37	16	14	30	10	11	21
Cardiovascular	21	16	37	22	15	37	11	12	23	30	29	59
Gastrointestinal	7	4	11	8	9	17	10	6	16	7	8	15
Urogenital	28	2	30	31	5	36	22	1	23	14	4	18
Musculoskeletal	18	31	49	35	34	69	16	30	46	27	28	55
Mouth and upper airways	6	11	17	8	5	13	14	8	22	6	12	18
Eye and ear	4	2	6	2	0	2	3	0	3	0	0	0
Chest	1	2	3	1	0	1	0	0	0	0	1	1
Syndromes and other defects	1	5	6	6	7	13	10	6	16	5	10	15
Total	85	74	159	109	84	193	92	71	163	87	83	170

TABLE 7. NUMBER OF MALFORMED CHILDREN OBSERVED IN SHAWINIGAN COMPARED WITH EXPECTED NUMBERS BASED ON RATES FOR THE COMPARISON COMMUNITIES (1966-1979)[a]

Affected System	Observed in Shawinigan	Expected Based on Drummondville	Expected Based on Baie-Comeau--Hauterive	Expected Based on Rimouski	Expected Based on All Comparison Communities
Central nervous	30	19.69*	18.18*	15.33**	17.96*
Cardiovascular	37	19.69**	13.94**	43.06	24.29*
Gastrointestinal	11	9.04	9.70	10.95	9.80
Urogenital	30	19.15*	13.94**	13.14**	15.72**
Musculoskeletal	49	36.71	27.88**	40.14	34.70*
Mouth and upper airways	17	6.92**	13.33	13.14	10.82
Eye and ear	6	1.06**	1.82*	--	1.02**
Chest	3	0.53*	--	0.73	0.41*
Syndromes and other defects	6	6.92	9.70	10.95	8.98
Total	159	102.68**	98.80**	124.08**	107.36**

[a]Ratio of Poisson variable to its expectation (36); * indicates $p < 0.05$; ** indicates $p < 0.01$.

children among the stillborn was higher in Shawinigan (27.3%) than in the comparison communities (total, 12.4%; Drummondville, 15.3%; Baie-Comeau--Hauterive, 10.0%; Rimouski, 10.2%; chi-square with Yates correction = 4.07, $p = 0.04$). This is consistent with previous findings of an excess of birth defects in Shawinigan.

Table 9 shows the distribution of children with multiple defects among the four communities under study. A chi-square test comparing the frequency of one birth defect versus multiple birth defects in Shawinigan and the three comparison communities indicates a higher proportion of multiple birth defects in Shawinigan ($X^2_1 = 4.18$, $p = 0.04$). However, this result was due to a low frequency of multiple birth defects in Baie-Comeau--Hauterive only.

Table 10 compares the distribution of birth defects for each year from 1966 to 1979 between Shawinigan and the other three communities. The proportion of malformed infants was greater in Shawinigan than in the comparison communities in every year except 1969 and 1972.

Table 11 compares the ages at delivery of Shawinigan mothers who gave birth to malformed children with the ages of mothers who gave birth to malformed children in the comparison communities. The mothers were significantly younger in Shawinigan for all malformations combined and for defects of the CNS, mouth and upper airways, and eye and ear.

TABLE 8. NUMBERS OF STILLBIRTHS, WITH AND WITHOUT BIRTH DEFECTS, AND NUMBERS EXPECTED IN SHAWINIGAN BASED ON RATES IN THE COMPARISON COMMUNITIES[a]

Condition	Number of Stillbirths			Total Number of Births
	With Birth Defects	Without Birth Defects	Total	
Observed in Shawinigan	9	24	33	4567
Observed in Drummondville	15	83	98	8584
Expected based on Drummondville	7.98	44.16**	52.14**	--
Observed in Baie-Comeau--Hauterive	7	63	70	7535
Expected based on Baie-Comeau--Hauterive	4.24	38.18*	42.42	--
Observed in Rimouski	5	44	49	6257
Expected based on Rimouski	3.65*	32.12	35.77	--
Expected based on all comparison communities	5.51	38.78	44.29	--

[a]Ratio of Poisson variable to its expectation (36); * indicates $p < 0.05$; ** indicates $p < 0.01$.

TABLE 9. DISTRIBUTION OF MALFORMED CHILDREN BY NUMBER OF BIRTH DEFECTS AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

Number of Birth Defects	Mother's Residence at Delivery			
	Shawinigan	Drummondville	Baie-Comeau--Hauterive	Rimouski
1	121	155	143	140
2	24	27	12	17
3	9	6	4	8
4	3	2	2	2
5	1	2	2	1
> 5	1	1	0	2
Total	159	193	163	170

TABLE 10. DISTRIBUTION OF MALFORMED CHILDREN FOR EACH YEAR (1966-1979)

Year	Shawinigan	Rate[a]	Comparison Communities	Rates	SH/Comparison Communities[b]	95% Confidence Interval
1966	16	3.54	31	1.82	1.95*	(1.08, 3.52)
1967	13	3.52	33	1.96	1.80	(0.96, 3.39)
1968	8	2.30	24	1.51	1.52	(0.69, 3.34)
1969	6	1.68	27	1.78	0.94	(0.41, 2.18)
1970	15	4.70	24	1.66	2.83*	(1.55, 5.17)
1971	6	2.08	30	2.05	1.01	(0.53, 1.94)
1972	6	2.19	34	2.43	0.90	(0.36, 2.20)
1973	18	6.84	44	3.17	2.16*	(1.28, 3.65)
1974	8	2.97	43	2.81	1.06	(0.48, 2.33)
1975	15	4.40	51	3.10	1.42	(0.81, 2.49)
1976	13	3.98	36	2.20	1.81	(0.93, 3.51)
1977	14	4.56	50	2.76	1.65	(0.93, 2.93)
1978	11	3.33	52	2.88	1.16	(0.61, 2.20)
1979	10	3.11	47	2.69	1.16	(0.60, 2.26)
Total	159	3.48	526	2.35	1.48*	(1.24, 1.75)

[a]Number of malformed children per 100 births. [b]* indicates $p < 0.05$.

TABLE 11. MEAN AGE OF THE MOTHER AT DELIVERY BY AFFECTED SYSTEM (1966-1979)

	Shawinigan			Comparison Communities			
Affected System	Mean Age	S.D.	N	Mean Age	S.D.	N	t-test[a]
Central nervous	24.63	3.44	30	27.97	6.78	86	2.58*
Cardiovascular	26.16	4.84	37	26.53	5.04	113	0.39
Gastrointestinal	27.64	5.33	11	27.72	5.94	46	0.04
Urogenital	24.90	4.01	30	26.07	4.85	72	1.16
Musculoskeletal	26.18	5.16	49	27.56	5.85	160	1.48
Mouth and upper airways	22.63	3.12	16	26.85	5.22	53	3.06**
Eye and ear	23.83	2.93	6	31.60	4.04	5	3.70**
Chest	22.33	1.15	3	26.00	2.83	2	2.13
Syndromes and other defects	27.50	4.51	6	30.07	6.47	41	0.94
Total	25.41	4.52	158[b]	27.38	5.73	498[b]	3.94***

[a]* indicates $p < 0.05$, ** indicates $p < 0.01$, and *** indicates $p < 0.001$.
[b]Age unknown for 1 mother in Shawinigan and 28 in the comparison communities.

Children in Shawinigan did not differ from those in the comparison communities in mean gestational age at birth, except in the case of chest malformation, for which the sample size was very small (Table 12).

TABLE 12. MEAN GESTATIONAL AGE AT BIRTH IN SHAWINIGAN AND THE COMPARISON COMMUNITIES, BY AFFECTED SYSTEM

	Shawinigan			Comparison Communities			
Affected System	Mean Gestational Age	S.D.	N	Mean Gestational Age	S.D.	N	t-test[a]
Central nervous	36.43	4.75	30	37.63	4.42	87	1.26
Cardiovascular	38.97	1.54	37	38.19	3.22	116	1.42
Gastrointestinal	38.45	1.86	11	38.13	3.21	47	0.32
Urogenital	37.77	4.01	30	38.77	2.94	73	1.40
Musculoskeletal	37.84	4.47	49	38.61	2.88	164	1.43
Mouth and upper airways	37.88	3.22	16	39.25	2.69	52	1.70
Eye and ear	38.83	2.48	6	37.40	3.97	5	0.73
Chest	38.67	1.53	3	33.00	1.41	2	4.17*
Syndromes and other defects	37.17	4.02	6	38.82	2.01	44	1.65
Total	38.32	3.38	158[b]	38.64	3.05	512[b]	1.12

[a]* indicates $p < 0.05$, ** indicates $p < 0.01$, and *** indicates $p < 0.001$.
[b]Mean gestational age unknown in 1 Shawinigan and 14 comparison-community cases.

SEASONAL VARIATION IN BIRTH-DEFECT RATES

Tables 13 and 14 give the monthly distribution of malformed children in Shawinigan, Drummondville, and Rimouski. Table 15 and Figure 1 compare the monthly birth-defect rates in Shawinigan (1966 through 1979) with those of two comparison communities (Drummondville and Rimouski, 1971 through 1977). The Shawinigan curve is V-shaped, with the lowest rates in the summer, whereas the curve for the comparison communities is flatter. Seasonal variation in birth-defect rates was compared between Shawinigan and the other three communities using Edward's method for testing seasonality of events, as modified by Walter (39) (see Table 16). There was significant seasonal variation in Shawinigan, with the highest rates in March and the lowest in September; no significant seasonal variation was observed in the comparison communities.

TABLE 13. DISTRIBUTION OF MALFORMED CHILDREN BY MONTH AND AFFECTED SYSTEM (SHAWINIGAN, 1966-1979)

Month of Birth	CNS	Cardio-vascular	Gastro-intes-tinal	Uro-genital	Musculo-skele-tal	Mouth & Upper Airways	Eye & Ear	Chest	Syndromes & Other Defects	Tot.
Jan.	3	6	1	4	3	1	0	0	1	17
Feb.	3	4	2	3	5	3	0	0	2	20
Mar.	5	4	0	0	3	1	1	0	0	13
Apr.	3	9	1	4	6	2	2	1	0	24
May	3	2	0	1	3	4	0	0	1	11
June	4	2	1	2	1	2	0	1	0	11
July	3	0	1	0	0	0	0	0	0	4
Aug.	0	3	1	1	6	2	2	0	0	11
Sep.	0	2	1	3	6	0	0	0	0	11
Oct.	1	0	0	3	9	0	0	1	1	15
Nov.	3	3	1	5	3	1	1	0	0	12
Dec.	1	2	2	3	3	1	0	0	1	9
Unkn.	1	0	0	1	1	0	0	0	0	1
Tot.	30	37	11	30	49	17	3	3	6	159

TABLE 14. DISTRIBUTION OF MALFORMED CHILDREN BY MONTH AND AFFECTED SYSTEM (DRUMMONDVILLE AND RIMOUSKI, 1971-1977)

Month of Birth	CNS	Cardio-vascular	Gastro-intes-tinal	Uro-genital	Musculo-skele-tal	Mouth & Upper Airways	Eye & Ear	Chest	Syndromes & Other Defects	Tot.
Jan.	0	5	3	1	3	0	0	1	0	9
Feb.	2	6	3	1	9	0	1	0	0	18
Mar.	3	6	0	5	6	3	0	0	1	23
Apr.	4	7	0	2	9	3	0	0	4	25
May	4	0	2	1	7	0	0	0	0	14
June	3	6	0	4	4	2	0	0	0	18
July	2	3	3	4	4	0	0	0	1	12
Aug.	3	7	1	2	5	2	0	0	3	20
Sep.	3	5	1	5	6	4	0	0	4	20
Oct.	3	4	2	2	5	3	0	0	1	20
Nov.	1	4	5	4	4	1	0	0	0	17
Dec.	2	5	0	4	4	1	0	1	1	10
Unkn.	0	1	0	0	0	0	0	0	0	1
Tot.	30	59	20	35	66	19	1	2	15	207

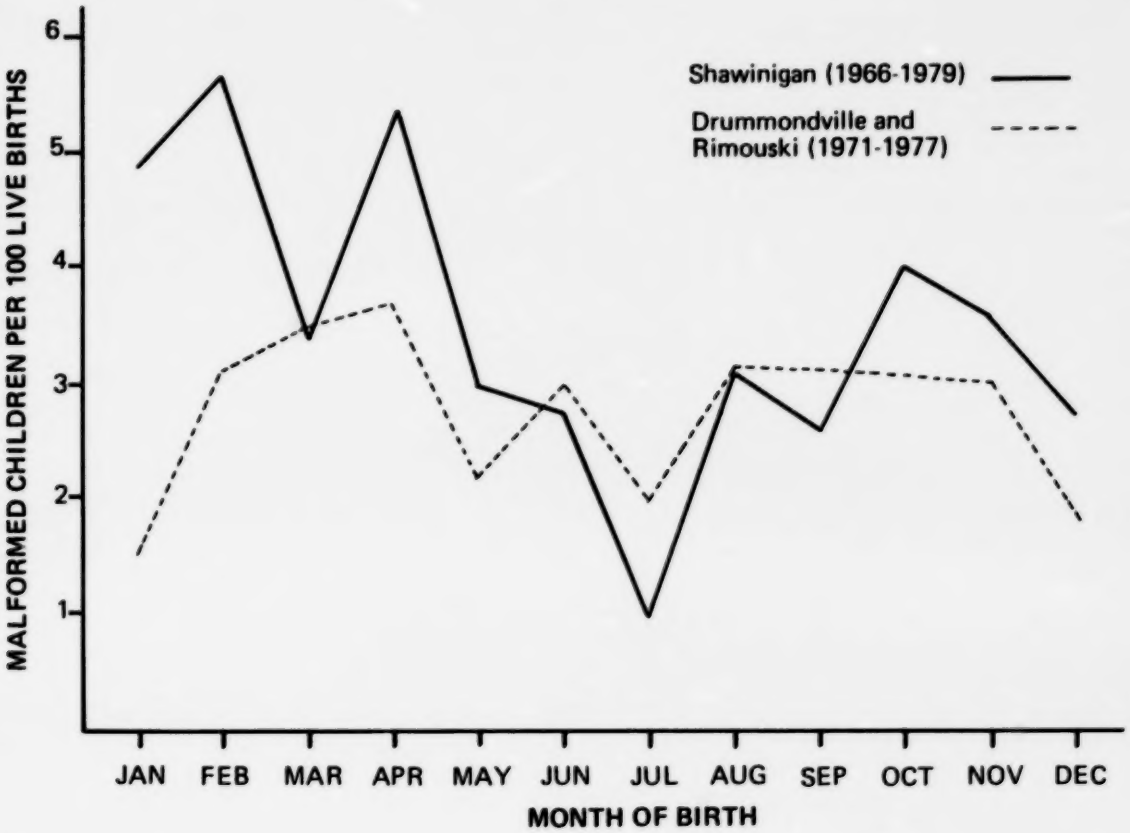


Figure 1. Monthly distribution of birth-defect rates.

SPATIAL VARIATION IN BIRTH-DEFECT RATES

The spatial distribution of birth defects with respect to the PVC polymerization plant was analyzed. Table 17 gives the distribution of malformed children by school district of the mother's residence and affected system. Table 18 gives the birth-defect rate for each school district. In no district did the ratio of observed to expected number of birth defects differ significantly from 1. District no. 14 had the highest rate, followed by nos. 8, 5, and 2. These districts are located on the north-south axis passing through the vinyl chloride plant; district nos. 14, 8, and 5 are north and district no. 2 is south of the plant. The plant is in district no. 3, which had about the expected number of birth defects.

TABLE 15. DISTRIBUTION OF MALFORMED CHILDREN FROM SHAWINIGAN AND THE COMPARISON COMMUNITIES BY MONTH OF BIRTH

Month of Birth	Shawinigan		Drummondville and Rimouski (1971-1977)	
	N	Rate[a]	N	Rate[a]
January	17	4.86	9	1.53
February	20	5.63	18	3.11
March	13	3.40	23	3.49
April	24	5.26	25	3.69
May	11	3.01	14	2.20
June	11	2.76	18	2.96
July	4	0.97	12	2.00
August	11	3.08	20	3.11
September	11	2.59	20	3.11
October	15	4.01	20	3.07
November	12	3.56	17	3.00
December	9	2.73	10	1.85
Unknown	1	--	1	--
Total	159	3.51	207	2.80

[a]Number of malformed children per 100 live births.

TABLE 16. ESTIMATES OF PARAMETERS OF SEASONAL VARIATION

Cities	Estimated Parameters[a]				
	d	o*	Month of Peak	X^2_2	p
Shawinigan	0.0742	59.48	March	7.005	< 0.02
Comparison communities	0.0185	-23.93	December	0.570	> 0.75

[a]Test for seasonality of events (39): d is the distance of the sample center of gravity from its null expectation, and o* is the direction of the maximum rate.

TABLE 17. DISTRIBUTION OF MALFORMED CHILDREN BY AFFECTED SYSTEM AND SCHOOL DISTRICT OF MOTHER'S RESIDENCE AT DELIVERY (SHAWINIGAN, 1966-1979)

School District	Affected System									
	CNS	Cardio-vascular	Gastro-intes-tinal	Uro-genital	Musculo-skele-tal	Mouth & Upper Airways	Eye & Ear	Chest	Syndromes & Other Defects	Tot.
1	2	1	1	1	2	1	0	0	0	5
2	1	3	0	0	2	1	0	0	1	7
3	2	0	0	0	4	0	0	1	0	6
4	0	0	0	4	2	1	0	0	0	7
5	2	4	4	7	4	3	1	0	1	19
6	2	4	1	4	5	3	1	0	0	18
7	3	1	1	5	6	1	2	0	0	14
8	5	7	1	3	8	2	1	0	1	25
9	1	1	1	2	1	1	0	1	0	8
10	3	2	0	0	3	1	0	0	2	9
11	2	3	0	0	2	0	0	0	0	7
12	0	2	1	0	1	0	0	1	1	5
13	0	4	0	1	4	0	1	0	0	10
14	6	4	1	2	4	3	0	0	0	17
Unkn.	1	1	0	1	1	0	0	0	0	2
Total	30	37	11	30	49	17	6	3	6	159

The yearly average concentration of vinyl chloride was estimated for each school district, based on data from a study entitled "Dispersion Patterns of Vinyl Chloride Emitted by B.F. Goodrich Co., Ltd., Shawinigan, Que." (which appears below as Appendix D). No differences in birth-defect rates were found between school districts with high and low VCM exposure, neither for all birth defects nor for CNS defects (see Table 19). Likewise, no differences were found in numbers of all birth defects or of CNS defects between the school districts adjacent to the plant and the other school districts (Table 20) or between school districts within and beyond a one-mile radius of the vinyl chloride plant (Table 21).

TABLE 18. DISTRIBUTIONS OF MALFORMED CHILDREN AND BIRTH-DEFECT RATES BY SCHOOL DISTRICT OF MOTHER'S RESIDENCE AT DELIVERY (SHAWINIGAN, 1966-1979)

School District	Observed N	Rate[a]	Expected N	Obs./Exp.
1	5	18.2	9.6	0.52
2	7	46.4	5.3	1.32
3	6	33.5	6.2	0.97
4	7	25.4	9.6	0.73
5	19	49.1	13.5	1.41
6	18	34.4	18.2	0.99
7	14	27.5	17.7	0.79
8	25	50.2	17.3	1.45
9	8	31.7	8.8	0.91
10	9	23.2	13.5	0.67
11	7	36.1	6.8	1.03
12	5	19.6	8.9	0.56
13	10	25.4	13.7	0.73
14	17	59.6	9.9	1.72
Unknown	2			
Total	159	34.8		

[a]Number of malformed children per 1000 live births.
[b]Applying the overall birth-defect rate to the number of live births in each school district.

TABLE 19. COMPARISON OF TOTAL BIRTH DEFECTS[a] AND CNS BIRTH DEFECTS[b] BETWEEN SCHOOL DISTRICTS WITH HIGH AND LOW ATMOSPHERIC VINYL CHLORIDE CONCENTRATION

	School Districts With High VCM Levels[c]	School Districts With Low VCM Levels	Total
Births with defects	87	70	157
Births without defects	2285	2125	4410
Births with CNS defects	16	13	29
Births without CNS defects	2356	2182	4538
Total births	2372	2195	4567

[a]$X^2_1 = 0.80$; $p > 0.35$. [b]$X^2_1 = 0.10$; $p > 0.70$.
[c]School districts nos. 1, 3, 5, 6, 7, and 8.

TABLE 20. COMPARISON OF TOTAL BIRTH DEFECTS[a] AND CNS BIRTH DEFECTS[b] BETWEEN SCHOOL DISTRICTS ADJACENT TO THE VINYL CHLORIDE PLANT AND THE OTHER SCHOOL DISTRICTS

	School Districts Adjacent to the Plant[c]	Other School Districts	Total
Births with defects	37	120	157
Births without defects	1080	3330	4410
Births with CNS defects	6	23	29
Births without CNS defects	1111	3427	4538
Total births	1117	3450	4567

[a] $X^2_1 = 0.07$; $p > 0.75$.
[b] $X^2_1 = 0.22$; $p > 0.60$.
[c] School districts nos. 1, 3, 4, and 5.

TABLE 21. COMPARISON OF TOTAL BIRTH DEFECTS[a] AND CNS BIRTH DEFECTS[b] BETWEEN SCHOOL DISTRICTS LOCATED INSIDE AND OUTSIDE A ONE-MILE RADIUS OF THE VINYL CHLORIDE PLANT

	Districts Within 1-Mile Radius[c]	Districts Outside 1-Mile Radius	Total
Births with defects	101	56	157
Births without defects	2698	1712	4410
Births with CNS defects	17	12	29
Births without CNS defects	2782	1756	4538
Total births	2799	1768	4567

[a] $X^2_1 = 0.64$; $p > 0.40$.
[b] $X^2_1 = 0.08$; $p > 0.75$.
[c] School districts nos. 1 through 8.

CASE-CONTROL STUDY

From January 1973 to December 1979, 89 cases of birth defects were identified in Shawinigan and matched with controls. Twenty-two pairs were lost for the following reasons: 4 cases and 2 controls were untraceable, 4 case mothers refused to be interviewed, 2 pairs were mismatched (1 for the sex of the children and 1 for the mother's age), 6 case mothers resided outside Shawinigan during the first third of their pregnancies, and 4 cases were diagnosed too late in 1979 to be included. One case from Baie de Shawinigan was found and added because this district is close to the VCM plant. Thus, there were 68 case-control pairs in the study (42 male and 26 female). Five of the cases were stillborn; all of the controls were born alive.

Table 22 lists the types of malformation presented by the 68 cases. Table 23 gives the distribution of cases and controls by the mother's age at delivery, which was matched within ± 2 years. There was no significant difference between cases and controls in number of weeks' gestation at delivery (see Table 24).

Previous birth defects in the families of cases and controls are listed in Table 25. Although more birth defects were reported in the cases' families, none of the differences between the groups were significant, nor was there a significant difference between cases and controls in number of previous pregnancies (see Table 26). Table 27 lists the diseases suffered during pregnancy by the mothers of cases and controls. There was no excess of diseases reported by the mothers of malformed infants. However, the following diseases suspected to bear a high risk of birth defect were reported more frequently by these than by the control mothers: rubella, hydramnios, epilepsy, nonpsychotic mental disorders, and psychosis. Among the mothers of malformed infants, five reported previous occurrences of epilepsy, mental disorders, or both. Their children showed the following birth defects: two cases of hypospadias, one of congenital cataract, one of Down's syndrome, and one interventricular septal defect. There was no significant difference between case and control mothers in number of previous abortions (see Table 28). There was one previous stillbirth among the control mothers and none for the case mothers; there was one previous malformed child in each of the two groups. As seen in Table 29, there was no significant difference between cases and controls in the father's age. No mother (case or control) was exposed to X-rays during the first trimester of pregnancy, and only one case mother was exposed during the second third of pregnancy. During the third trimester, 24 case mothers and 23 control mothers were exposed; this difference was not statistically significant ($X^2_1 = 0.13$; $p > 0.7$).

Table 30 summarizes the mothers' occupations before and during their pregnancies. None of the women were ever exposed to VCM at work before or during pregnancy. The working experiences of the case and control mothers before and during pregnancy did not differ, nor did their smoking habits (see Table 31). Four case and two control mothers consumed alcohol regularly during pregnancy; this difference was not significant ($X^2_1 = 0.70$, $p > 0.40$).

TABLE 22. DISTRIBUTION OF SHAWINIGAN CASES BY TYPE OF BIRTH DEFECT

Affected System	Male	Female	Description
Central nervous	6	2	anencephaly (1), hydrocephalus (6), encephalocele (3)
Cardiovascular	10	8	hypoplasia of the heart (1), atrioventricular canal defect (1), patent ductus arteriosis (1), cardiac valve anomalies (1), pulmonary stenosis (1), cor uniloculare (1), ventricular septal defect (13), heart murmurs (1)
Urogenital	14	-	hypospadias (14)
Musculoskeletal	8	9	syndactylia (3), polydactylia (1), clubfoot (5), defect in foot positioning (3), hip dislocation (4), congenital torticollis (1), facial asymmetry (1), deformity of the thorax (1)
Mouth and upper airways	1	5	cleft palate or cleft lip (3), congenital laryngeal stridor (3)
Eye and ear	1	2	congenital cataract (2), defect of the external auditory canal (1)
Syndromes and other defects	1	3	Down's syndrome (1), trisomy syndrome (1), congenital lymphedema (1), multiple birth defects (1)
Total	39	27	

TABLE 23. DISTRIBUTION OF CASES AND CONTROLS BY MOTHER'S AGE AT DELIVERY

Age of the Mother	Cases	Controls
< 20	4	4
20 - 24	31	31
25 - 29	19	22
30 - 34	12	9
35 - 39	2	2
Total	68	68

TABLE 24. DISTRIBUTION OF CASES AND CONTROLS BY WEEKS OF GESTATION[a]

Weeks of Gestation	Cases	Controls
30 - 34	1	1
35 - 36	5	4
37 - 38	12	11
39 - 40	41	37
41 - 42	9	14
Unknown	0	1
Total	68	68

[a]$X^2_4 = 1.45$; $p > 0.83$.

TABLE 25. DISTRIBUTION OF CASES AND CONTROLS BY PREVIOUS OCCURRENCE OF BIRTH DEFECTS IN THE FAMILY

Previous Occurrence of Birth Defects in the Family		Cases	Controls	RR[a]	95% Confidence Interval
Yes	on father's side	25	18	1.54	0.69, 3.63
	on mother's side	32	31	1.07	0.51, 2.24
	on both sides	11	13	0.78	0.11, 5.47
	total	46	36	1.83	0.85, 3.94
No		22	32		
Total		68	68		

[a]Based on MacNemar matched-pairs test.

Two mothers of defective infants reported having used the drug LSD before their pregnancies. There was no significant difference between cases and controls in the father's occupational exposure to chemicals, and no fathers were ever exposed to vinyl chloride (see Table 32).

Table 33 and Figure 2 shows the mother's place of residence during the first third of pregnancy. The distance between each place of residence and the VCM plant was measured to ± 100 m, using the map of the city. Wilcoxon's signed-ranks test (38) revealed no significant difference between the two groups in distance from the VCM plant.

TABLE 26. DISTRIBUTION OF CASES AND CONTROLS BY NUMBER OF PREVIOUS PREGNANCIES[a]

No. of Pregnancies	Cases	Controls
0	35	36
1	18	16
2	8	8
3	4	2
> 4	3	6
Total	68	68

[a] $X^2_4 = 1.80$; $p > 0.77$.

TABLE 27. DISTRIBUTION OF CASES AND CONTROLS BY DISEASES OF THE MOTHERS DURING PREGNANCY

Disease	Cases	Controls
None	44	48
Bleeding at the beginning of the pregnancy	6	3
Incoercible vomiting	1	1
Hypertension	3	6
Rubella	1	0
Lithiasis of the gall bladder	1	1
Renal calculus	1	0
Psychosis	1	0
Premature labor	2	1
Phlebitis	1	0
Hydramnios	2	0
Obesity	0	3
Placenta praevia	0	1
Epilepsy	1	0
Nonpsychotic mental disorders	2	0
Wound	0	1
Infectious or parasitic disease	4	6
Other diseases of the mother	3	2
Total for diseases	24	20
Unknown	0	0
Total	68	68

[a] Partitioning diseases vs. no disease, $X^2_1 = 0.54$; $p > 0.45$.

TABLE 28. DISTRIBUTION OF CASES AND CONTROLS BY NUMBER OF PREVIOUS ABORTIONS (AS RECORDED IN HOSPITAL MEDICAL FILES)[a]

Number of Previous Abortions	Cases	Controls
0	55	60
1	11	5
2	1	1
3	1	0
> 4	0	2
Total	68	68

[a]Partitioning none vs. one or multiple abortions, $X^2_1 = 1.41$; $p > 0.23$.

TABLE 29. DISTRIBUTION OF CASES AND CONTROLS BY FATHER'S AGE AT BIRTH OF MALFORMED INFANT[a]

Father's Age	Cases	Controls
< 19	1	2
20 - 23	15	4
24 - 27	18	24
28 - 31	17	22
32 - 35	12	10
36 - 39	4	3
> 40	0	2
Unknown	1	1
Mean age	27.66	28.66
Standard deviation	4.52	4.96

[a]t-test: $t = 1.23$; $p > 0.05$.

TABLE 30. DISTRIBUTION OF CASES AND CONTROLS BY MOTHER'S OCCUPATION[a]

Mother's Occupation	Cases		Control	
	Before Pregnancy	During Pregnancy	Before Pregnancy	During Pregnancy
Work outside home without exposure to chemicals	41	22	42	20
Work in VCM industry	0	0	0	0
Work outside home with exposure to chemicals	2	1	2	1
Stay at home	25	45	24	47
Total	68	68	68	68

[a]Partitioning stay at home vs. work outside home before pregnancy: $X^2_1 = 0.03$; $p > 0.85$. Partitioning stay at home vs. work outside home during pregnancy: $X^2_1 = 0.13$; $p > 0.71$.

TABLE 31. DISTRIBUTION OF CASES AND CONTROLS BY SMOKING HABITS OF THE MOTHER[a]

Smoking Habits of the Mother	Cases		Control	
	Before Pregnancy	During Pregnancy	Before Pregnancy	During Pregnancy
Smoker	34	26	41	32
Ex-smoker	3	11	2	11
Nonsmoker	31	31	25	25
Total	68	68	68	68

[a]For smokers vs. ex- and nonsmokers before pregnancy, $X^2_1 = 1.46$; $p > 0.20$. For smokers vs. ex- and nonsmokers during pregnancy, $X^2_1 = 1.08$; $p > 0.25$.

TABLE 32. DISTRIBUTION OF CASES AND CONTROLS BY FATHER'S OCCUPATION[a]

Father's Occupation	Cases	Controls
Ever worked in vinyl chloride industry	0	0
Ever worked in industries with exposure to chemicals	20	25
Never worked in industries with exposure to chemicals	43	39
Unknown	5	4
Total	68	68

[a] $X^2_1 = 0.73$; $p > 0.39$.

TABLE 33. DISTRIBUTION OF CASES AND CONTROLS BY MOTHER'S RESIDENCE DURING THE FIRST THIRD OF PREGNANCY

School District	Cases	Controls
1	1	3
2	5	2
3	1	2
4	3	6
5	7	7
6	8	8
7	3	7
8	13	8
9	4	1
10	3	9
11	2	0
12	1	2
13	8	8
14	7	3
Baie de Shawinigan	1	2
Unknown	1	0
Total	68	68

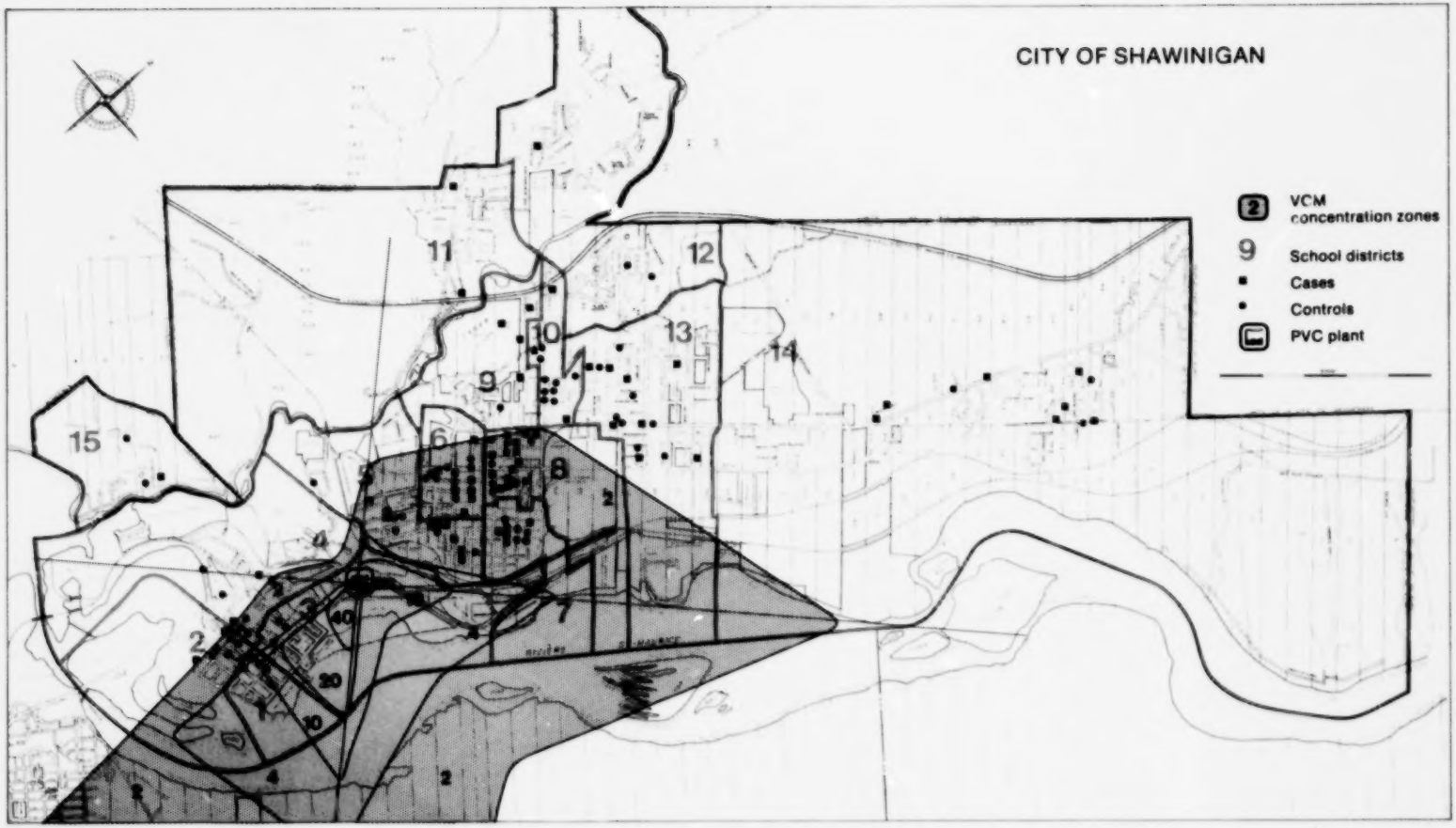


Figure 2. Residences of birth-defect cases and controls at conception and distribution of VCM concentration.

SECTION 5

DISCUSSION

PREVIOUS STUDIES

Excesses of birth defects in communities in the vicinity of vinyl chloride polymerization plants and the possible association between these birth defects and emissions of vinyl chloride monomer into the atmosphere were first reported for three Ohio communities (28). This study revealed that between 1970 and 1973, birth defects were more frequent in Ashtabula, Painesville, and Avon Lake, where PVC plants operated, than in the state of Ohio or in the balance of the three counties where these cities were located. The excess was constant throughout the study. Mothers who gave birth to malformed children were, on average, one year younger in the index communities than in the balance of the three counties. The excess in birth defects involved mainly defects of the central nervous system (CNS), upper alimentary tract, and genital organs, and clubfoot. Cleft palate and cleft lip, as well as several other malformations, were also in excess, although not significantly so.

In analyzing data collected through the Center for Disease Control's hospital-based birth defects monitoring program, another investigator (29) found no increase in CNS defects for one city in Pennsylvania with a PVC plant, but confirmed the excess of anencephaly and spina bifida previously noted in Painesville, Ohio. However, in a case-control study, it was found that none of the interviewed parents of affected infants had ever worked at either of the two PVC polymerization plants in Painesville, whereas two of the fathers of controls did; none of the parents in either group lived within two miles of either plant. Furthermore, a significantly larger proportion of control mothers than case mothers worked within a ten-mile radius of the PVC plant. Although a moderate excess of CNS malformations in Painesville was confirmed, it was not found to be associated with exposure of the parents to vinyl chloride.

A subsequent study (23) emphasized not the presence of birth defects among infants whose mothers were exposed to VCM during pregnancy but rather fetal losses and miscarriages among women whose husbands worked at PVC plants. Although it was concluded that fetal losses were greater among women whose husbands were occupationally exposed to VCM than among controls, this study was criticized for methodological weaknesses.

In a report to the Society of Plastics Industries, MacMahon (24) concluded that there was good evidence only for chromosome breakage in heavily exposed VCM workers, but that VCM should be regarded as a potential human mutagen and

teratogen until better evidence was available. In another report to the Society, Downs et al. (25) criticized the statistical methods used in vinyl chloride studies, concluding that the existing evidence indicated associations between 1) VCM exposure and lymphocyte aberrations in workers and 2) VCM exposure of workers and fetal wastage among the workers' wives. These authors found no evidence for an association between VCM exposure and birth defects, but cautioned that such associations may not have been detectable by methods available at the time and that monitoring should continue.

A high incidence of CNS birth defects was found in Kanawha Co., West Virginia, from 1970 to 1974 (30). As there is a polyvinyl chloride plant in Kanawha Co., a possible relationship between CNS defects and parental exposure to VCM was investigated. In a case-control study, no relationship was found between birth defects and parental occupation. Although the pattern of case-family residences tended to differ from that of controls, it did not correlate with local wind-direction and air-pollution patterns.

THE PRESENT STUDY

Some observations of the present study support the existence of an association between VCM in the air and birth defects in the exposed community, while other observations tend to contradict such an association. High birth-defect rates in Shawinigan, observed in a previous study (31), were confirmed for a 15-year period by comparison with three other communities lacking PVC plants. This is consistent with the findings for three Ohio towns with vinyl chloride plants. Furthermore, this study revealed seasonal variation in birth-defect rates in Shawinigan that could correspond to variation in atmospheric VCM concentrations. VCM could not be measured in air samples during December, January, and February (see Appendix C). It is believed that during these months, VCM tends to accumulate near the plant as a liquid, because the outdoor temperature is below its liquefaction point (-13°C). This VCM would be released into the air in the spring. Birth-defect rates were lowest in September, which is eight months after midwinter, corresponding to the time between the first third of pregnancy and delivery. This observation would tend to support an association between VCM concentration in the air and birth defects in the community. One consultant believed that below its liquefaction point, VCM would remain in the air in droplets, at a concentration similar to that found at higher temperatures. However, because people spend more time indoors during the winter than the rest of the year, winter exposure would tend to be low in any case.

Another observation of the present study was that the women from Shawinigan who gave birth to malformed children were, on average, younger than women from the comparison communities who gave birth to malformed infants. This phenomenon could be a result of an association between VCM and birth defects. Similar results were found for the three cities in Ohio discussed above (28).

On the other hand, several observations of the present study tend to argue against any association between VCM concentration in the air and birth defects in the community. The first is the spatial distribution of birth

defects in Shawinigan, which cannot be explained in any logical way on the basis of estimates of VCM concentrations in the community. Malformation rates were not relatively high either near the plant or in the area where VCM concentrations were estimated to be highest. The school district with the highest birth-defect rate was far from the plant, where there was no reason to believe that VCM concentrations were high. However, VCM concentrations were not measured directly, but estimated by a theoretical dispersion model (see Appendix D) based on approximate production and emission values. Actual values would have been more reliable, but the B.F. Goodrich Co. would not release this information.

The second reason to believe that there was no association between VCM in the air and birth defects in the community is the lack of any difference in occupational or residential history between the parents who gave birth to malformed infants and the control parents. Both groups resided at similar distances from the plant, and none of the parents had worked at the plant. The third observation that argues against an association between VCM and birth defects is that in Shawinigan, all types of birth defects were in excess, rather than those of any particular system. Although these results agree with those for the three Ohio cities (28), agents known to be teratogenic in humans and experimental animals have very specific effects. It seems unlikely that any agent would produce the variety of malformations found in excess in Shawinigan. One must therefore search for artifacts that could account for the observed excess in birth defects.

One such artifact could be that Shawinigan's physicians were more inclined to diagnose birth defects than physicians in the comparison communities. There are several reasons that this is unlikely: birth defects included in this study were selected on the basis of their obviousness; furthermore, the birth-defect rates were the same in the three comparison communities, whereas a bias based on physicians' diagnoses would have generated more variation in rates among the communities; moreover, for severe birth defects like those of the CNS, which cannot be misdiagnosed, rates were much higher in Shawinigan than in any of the comparison communities.

Another source of artifact could involve the quality of the information from the archives of the regional hospitals. The method by which the data was collected made it very unlikely that such a bias could have influenced the results. In Shawinigan and Drummondville, the data were collected from a list of birth-defect cases provided by the hospital, while in Baie-Comeau--Hauterive and Rimouski, the medical records of all newborns were reviewed to identify malformed children. On analysis, no difference in birth-defect rates was found among the three comparison communities. Moreover, quality-control procedures, described in the Methods section of this report, were followed in data collection.

The possibility remains that pregnant mothers residing outside of Shawinigan who were at risk of having a malformed child migrated to the city for delivery, thereby increasing the birth-defect rate for that town. This possibility deserves special attention. In the case-control study, six mothers (6.7%) were rejected because their addresses during the first third of pregnancy were outside the city of Shawinigan, although their addresses at

delivery were in Shawinigan. Four more cases were untraceable. This could mean that as many as 11% of the birth-defect cases observed in Shawinigan actually came from elsewhere, reducing the number of observed cases to 142. However, birth-defect rates would still be significantly higher than in the comparison communities, leading us to believe that the excess noted in Shawinigan was real.

Animal studies have revealed that exposure to high levels of VCM during pregnancy generates signs of embryotoxicity, manifested more often by high rates of fetal loss and miscarriage than by birth defects in the offspring (26, 27). Stillbirth rates were no higher in Shawinigan than in the comparison communities. Furthermore, in the case-control study, abortion rates were no higher for case than for control mothers. This observation tends to indicate that VCM in the air did not affect fetuses.

Several industries in Shawinigan emit pollutants into the atmosphere: an aluminum electrolysis plant, a paper mill, and several chemical plants. Several of the emitted pollutants may interact to generate potent mutagenic or teratogenic chemicals in the community. The analysis of such an environment and its association with the excess of birth defects was beyond the scope of the present study. It should be noted, however, that in the case-control study, there was no difference between the numbers of case and control parents who worked at the aluminum plant. Furthermore, the comparison community with an aluminum electrolysis plant (Baie-Comeau--Hauterive) had lower birth-defect rates than did Shawinigan. It is thus questionable that the aluminum industry alone was responsible for the excess of birth defects in Shawinigan.

Several authors have noted sex differences and trends of annual decreases in various types of birth defects (32, 33). No such trends or differences were noted in this study.

This study investigated several risk factors, other than occupational or environmental exposure to a pollutant, known to be associated to some degree with birth defects. These factors included maternal and paternal ages at delivery; family history of birth defects; previous pregnancies, abortions, stillbirths, and malformed children; previous diseases; diseases during pregnancy; maternal exposure to X-rays during pregnancy; and alcohol, cigarette, and drug consumption before and during pregnancy. Epilepsy and mental disorders before and during pregnancy and the use of the drug lysergic acid diethylamide (LSD) before pregnancy were reported more frequently by mothers of malformed children than by control mothers. Drugs prescribed to control epilepsy have been reported to be associated with birth defects (40) and could account for a few birth defects observed in Shawinigan, as could LSD. No other risk factor was reported more frequently by the cases than by the controls. Other factors, such as the environment, remain to be studied.

In summary, some observations of the present study point toward an association between VCM in the air and birth defects in the exposed community, whereas others are evidence against such an association. We conclude that such an association cannot be substantiated at the present time and that, if it exists, it cannot be measured by the methods used in this and in previous studies or in populations of the sizes studied so far.

REFERENCES

1. Viola, P.L., A. Bigotti, and A. Caputo. Oncogenic response of rat skin, lungs, and bones to vinyl chloride. Cancer Res. 31:516-522, 1971.

2. Maltoni, C., and G. Lefemine. Carcinogenicity bioassays of vinyl chloride: research plans and early results. Environ. Res. 7:387-405, 1974.

3. Maltoni, C., and G. Lefemine. Carcinogenicity bioassays of vinyl choride: current results. Ann. N.Y. Acad. Sci. 246:195-218, 1975.

4. Creech, J.L., Jr., and M.N. Johnson. Angiosarcoma of liver in the manufacture of polyvinyl chloride. J. Occup. Med. 16:150-151, 1974.

5. Thomas, L.B., and H. Popper. Pathology of angiosarcoma of the liver among vinyl chloride-polyvinyl chloride workers. Ann. N.Y. Acad. Sci. 246:268-277, 1975.

6. Makk, L., F. Delorme, J.L. Creech, L.L. Ogden, E.H. Fadell, C.L. Songster, J. Clanton, M.N. Johnson, and W.M. Christopherson. Clinical and morphologic features of hepatic angiosarcoma in vinyl chloride workers. Cancer 37:149-163, 1976.

7. Delorme, F., and G. Thériault. Ten cases of angiosarcoma of the liver in Shawinigan, Quebec. J. Occup. Med. 20:338-340, 1978.

8. Monson, R.R, J.M. Peters, and M.N. Johnson. Proportional mortality among vinyl chloride workers. Lancet ii:397-398, 1974.

9. Waxweiler, R.J., W. Stringer, J.K. Wagoner, J. Jones, H. Falk, and C. Carter. Neoplastic risk among workers exposed to vinyl chloride. Ann. N.Y. Acad. Sci. 271:40-48, 1976.

10. Funes-Cravioto, F., B. Lambert, J. Lindsten, L. Ehrenberg, A.I. Natarjan, and S. Osterman-Golkar. Chromosome aberrations in workers exposed to vinyl chloride. Lancet i:459, 1975.

11. Ducatman, A., K. Hirschhorn, and I.J. Selikoff. Vinyl chloride exposure and human chromosome aberrations. Mutation Res. 31:163-168, 1975.

12. Fleig, I., and A.M. Thiess. Mutagenicity of vinyl chloride. J. Occup. Med. 20:557-561, 1978.

13. Léonard, A., G. Decat, E.D. Léonard, M.J. Lefèvre, L.J. Decuyper, and C. Nicaise. Cytogenetic investigations on lymphocytes from workers exposed to vinyl chloride. J. Toxicol. Environ. Hlth. 2:1135-1141, 1977.

14. Hansteen, I.-L., L. Hillestad, E. Thiis-Evensen, and S.S. Heldaas. Effects of vinyl chloride in man, a cytogenetic follow-up study. Mutation Res. 51:271-278, 1978.

15. Kučerová, M., Z. Polívková, and J. Bátora. Comparative evaluation of the frequency of chromosomal aberrations and the SCE numbers in peripheral lymphocytes of workers occupationally exposed to vinyl chloride monomer. Mutation Res. 67:97-100, 1979.

16. Drevon, C., and T. Kuroki. Mutagenicity of vinyl chloride, vinylidene chloride and chloroprene in V79 Chinese hamster cells. Mutation Res. 67:173-182, 1979.

17. Bartsch, H., and R. Montesano. Mutagenic and carcinogenic effects of vinyl chloride. Mutation Res. 32:93-113, 1975.

18. Loprieno, N., R. Barale, S. Baroncelli, C. Bauer, G. Bronzetti, A. Cammellini, G. Cercignani, C. Corsi, G. Gervasi, C. Leporini, R. Nieri, A.M. Rossi, G. Stretti, and G. Turchi. Evaluation of the genetic effects induced by vinyl chloride monomer (VCM) under mammalian metabolic activation: studies in vitro and in vivo. Mutation Res. 40:85-95, 1976.

19. Magnusson, J., and C. Ramel. Mutagenic effects of vinyl chloride in Drosophila melanogaster. Mutation Res. 38:115 (abstract), 1976.

20. Vainio, H. Vinyl chloride and vinyl benzene (styrene)--metabolism, mutagenicity and carcinogenicity. Chem.-Biol. Interact. 22:117-124, 1978.

21. Rannug, U., A. Johansson, C. Ramel, and C.A. Wachtmeister. The mutagenicity of vinyl chloride after metabolic activation. Ambio 3:194-197, 1974.

22. Green, T., and D.E. Hathway. Interactions of vinyl chloride with rat-liver DNA in vivo. Chem.-Biol. Interact. 22:211-224, 1978.

23. Infante, P.F., J.K. Wagoner, A.J. McMichael, R.J. Waxweiler, and H. Falk. Genetic risks of vinyl chloride. Lancet i:734-5, 1976.

24. MacMahon, B. Vinyl Chloride and Human Reproduction. Manuscript prepared for the Society of Plastics Industries, Inc., Aug. 11, 1977.

25. Downs, T.D., R.A. Stallones, R.F. Frankowski, and D.R. Labarthe. Vinyl Chloride, Birth Defects, and Fetal Wastage: A Critical Review. Prepared for the Society of Plastics Industries, Inc., Sept. 16, 1977.

26. Ungváry, G., A. Hudák, E. Tátrai, M. Lörincz, and G. Folly. Effects of vinyl chloride exposure alone and in combination with trypan blue--applied systematically during all thirds of pregnancy on the fetuses of CFY rats. Toxicol. 11:45-54, 1978.

27. John, J.A., F.A. Smith, B.K.J. Leong, and B.A. Schwetz. The effects of maternally inhaled vinyl chloride on embryonal and fetal development in mice, rats, and rabbits. Toxicol. Appl. Pharmacol. 39:497-513, 1977.

28. Infante, P.F. Oncogenic and mutagenic risks in communities with polyvinyl chloride production facilities. Ann. N.Y. Acad. Sci. 271:49-57, 1976.

29. Edmonds, L.D., H. Falk, and J.E. Nissim. Congenital malformations and vinyl chloride. Lancet ii:1098, 1975.

30. Edmonds, L.D., C.E. Anderson, J.W. Flynt, Jr., and L.M. James. Congenital central nervous system malformations and vinyl chloride monomer exposure: a community study. Teratol. 17:137-142, 1978.

31. Thériault, G.P., and L. Goulet. Birth defects in a community located near a vinyl chloride plant. Unpublished manuscript.

32. Myrianthopoulos, N.C., and C.S. Chung. Congenital malformations in singletons: epidemiologic survey. Birth Defects 10(11):1-58, 1974.

33. Leck, I. The etiology of human malformations: insights from epidemiology. Teratology 5:303-314, 1972.

34. Snedecor, G.W., and W.G. Cochran. Statistical Methods. Iowa State University Press, Ames, 593 pp., 1967.

35. Neter, J., and W. Wasserman. Applied Linear Statistical Models. Richard D. Irwin, Inc., Homeward, IL, 1977.

36. Bailar, J.C., and F. Ederer. Significance factors for the ratio of a Poisson variable to its expectation. Biometrics 20:639-643, 1964.

37. Miettinen, O.S. Simple interval-estimation of risk ratio. Am. J. Epidemiol. 100:515-516 (abstract), 1974.

38. Armitage, P. Statistical Methods in Medical Research. Blackwell Scientific Publications, Oxford, England, 1977.

39. Walter, S.D., and J.M. Elwood. A test for seasonality of events with a variable population at risk. Brit. J. Prevent. Soc. Med. 29:18-21, 1975.

40. Smithells, R.W. Environmental teratogens of man. Brit. Med. Bull. 32:27-33, 1976.

APPENDIX A

DETAILS OF METHODS

CONTENTS

		PAGE
Birth Defects Included in the Study		A-2
Table A-1.	Numbers of birth-defect cases found at hospitals visited	A-3
Table A-2.	Populations of towns, by sex and age groups, in 1971	A-4
Table A-3.	Average annual numbers of births and birthrates for 1966-1979	A-4
Table A-4.	Percentages of labor forces in various economic activities in 1971	A-5
Table A-5.	Principal industries of towns in 1978	A-5
Table A-6.	Average family incomes in 1971	A-6
Table A-7.	Ratios of physicians to population and hospital beds to population in 1978	A-6
Figure A-1.	Increment of the populations of females aged 15 to 44 years, from 1966 to 1976	A-7
Figure A-2.	Age and sex distributions of the populations of Rimouski and Baie-Comeau--Hauterive	A-8
Figure A-3.	Age and sex distributions of the populations of Shawinigan and Drummondville	A-9
Figure A-4.	Sample of questionnaire used in case-control study	A-10

BIRTH DEFECTS INCLUDED IN THE STUDY

Central nervous system defects and related skeletal conditions: anencephaly, craniosynostosis, bone defect of the skull, encephalocele, meningocele, hydrocephalus, and spina bifida.

Eye conditions: anophthalmus and congenital cataract.

Ear conditions: absence or deformity of the external meatus.

Upper respiratory tract and mouth conditions: cleft palate, cleft lip, choanal atresia, choanal stenosis, and absent uvula.

Thoracic abnormalities: pneumomediastinum, absence of the lung, pneumatocele, hiatal hernia, and thoracic deformity.

Cardiovascular conditions: cardiac englargement, hypoplasia of the heart, ventricular septal defect, patent or absent ductus arteriosus, valve defects, transposition of great vessels, coarctation of the aorta, cor biloculare-cor triloculare, and Fallot's tetralogy.

Digestive tract malformations: tracheoesophageal fistula, eosophageal atresia, pyloric stenosis, duodenal atresia, small intestine atresia, colon atresia, megacolon, Meckel's diverticulum, inguinal hernia, umbilical hernia, rectal fistula, imperforated anus, and liver and biliary duct defects.

Urogenital conditions: hypospadias, epispadias, penis hypoplasia and malformation, agenesis of the testicle, clitoral hypertrophy, internal genital malformation, hermaphroditism, and all kidney, ureter, bladder, and urethral malformations.

Musculoskeletal abnormalities: clubfoot, syndactyly, polydactyly, phocomelia, rib anomalies, dysplasia of the hip, congenital torticollis, hemihypertrophia, macrosomatia, and other foot abnormalities that required either a cast or surgery for correction.

Infection: congenital rubeola syndrome and congenital lues syndrome.

Syndromes: Down's syndrome and trisomia.

Other endocrine or metabolic conditions: cretinism and congenital goiter.

TABLE A-1. NUMBERS OF BIRTH-DEFECT CASES FOUND AT HOSPITALS VISITED

Hospital	Number of Birth-Defect Cases
Hôpital Ste-Croix de Drummondville	192
Hôpital de Chicoutimi	1
Hôpital de Jonquière	2
Centre hospitalier régional de la Mauricie	119
Hôpital Ste-Marie (Trois-Rivières)	3
Hôpital St-Joseph (Trois-Rivières)	2
Hôpital Laflèche (Grand-Mère)	33
Hôpital St-Joseph (Rimouski)	172
Hôpital général (Baie-Comeau)	85
Hôtel-Dieu de Hauterive	72
Hôpital St-François d'Assise (Québec)	3
Hôpital de la Miséricorde (Drummondville)	1
Hôpital Enfant-Jésus (Québec)	0
Hôpital St-Sacrement (Québec)	0
Hôpital Jeffery Hale (Québec)	0
Hôpital Cloutier (Cap-de-la-Madeleine)	0
Total	685

TABLE A-2. POPULATIONS OF TOWNS, BY SEX AND AGE GROUPS, 1971

Age Group	Sex	Shawinigan	Drummondville	Rimouski	Baie-Comeau--Hauterive
0-14	M	3,675	4,190	3,925	4,670
	F	3,480	4,105	3,860	4,640
	T	7,155	8,295	7,785	9,310
15-44	M	6,260	7,585	6,290	6,480
	F	6,325	7,635	7,135	6,300
	T	12,585	15,220	13,425	12,780
45-64	M	2,800	2,800	1,955	1,545
	F	3,160	3,240	2,270	1,295
	T	5,960	6,040	4,225	2,840
> 65	M	920	935	610	175
	F	1,175	1,315	850	195
	T	2,095	2,250	1,460	370
Total	M	13,655	15,510	12,780	12,870
	F	14,140	16,295	14,115	12,430
	T	27,795	31,805	26,895	25,300

TABLE A-3. AVERAGE ANNUAL NUMBERS OF BIRTHS AND BIRTHRATES FOR 1966-1979

	Shawinigan	Drummondville	Rimouski	Baie-Comeau--Hauterive
Number of births	325	541	444	548
per 1000	11.2	17.4	17.0	21.5
per 1000 women aged 15-44	51.4	70.9	62.2	87.0

TABLE A-4. PERCENTAGE OF LABOR FORCE IN VARIOUS ECONOMIC ACTIVITIES IN 1971, BY SEX

Economic Activity	Shawinigan		Drummondville		Rimouski		Baie-Comeau--Hauterive	
	M	F	M	F	M	F	M	F
Manufacturing	43.8	15.8	48.7	34.1	8.4	3.0	38.8	5.2
Transport and communications	7.6	9.8	6.2	2.8	16.0	10.6	16.2	2.5
Commercial	14.0	14.6	15.9	31.5	15.6	11.6	8.9	14.5
Services and administration	18.5	49.4	18.5	41.8	35.4	51.4	18.5	55.4

TABLE A-5. PRINCIPAL INDUSTRIES OF TOWNS IN 1978[a]

Town	Principal Industries	No. of Workers
Shawinigan	pulp and paper	1047
	aluminum	790
	cellulose film	474
	inorganic industrial chemicals	360
	textiles	219
	polyvinyl chloride	207
	abrasives	202
	caustic soda, chlorine, and chlorhydrocarbons	161
Drummondville	inorganic industrial chemicals	1468
	textiles	1157
	printing	1087
	fluorescent lamps, electronic tubes	687
Rimouski	steel structures	175
	foods	102
	lumber and furniture	93
	aluminum telephone and electrical cables	93
Baie-Comeau--Hauterive	pulp and paper	2868
	aluminum	790

[a]From Scott's Industrial Directory of Quebec.

TABLE A-6. AVERAGE FAMILY INCOMES IN 1971

Town	Average Income ($)
Shawinigan	7,881
Drummondville	8,652
Rimouski	9,228
Baie-Comeau--Hauterive	10,954

TABLE A-7. RATIOS OF PHYSICIANS TO POPULATION AND HOSPITAL BEDS TO POPULATION, IN 1978

Town	Physicians	Hospital Beds
Shawinigan	1:669	1:115
Drummondville	1:779	1:66
Rimouski	1:535	1:77
Baie-Comeau--Hauterive	1:1260	1:144

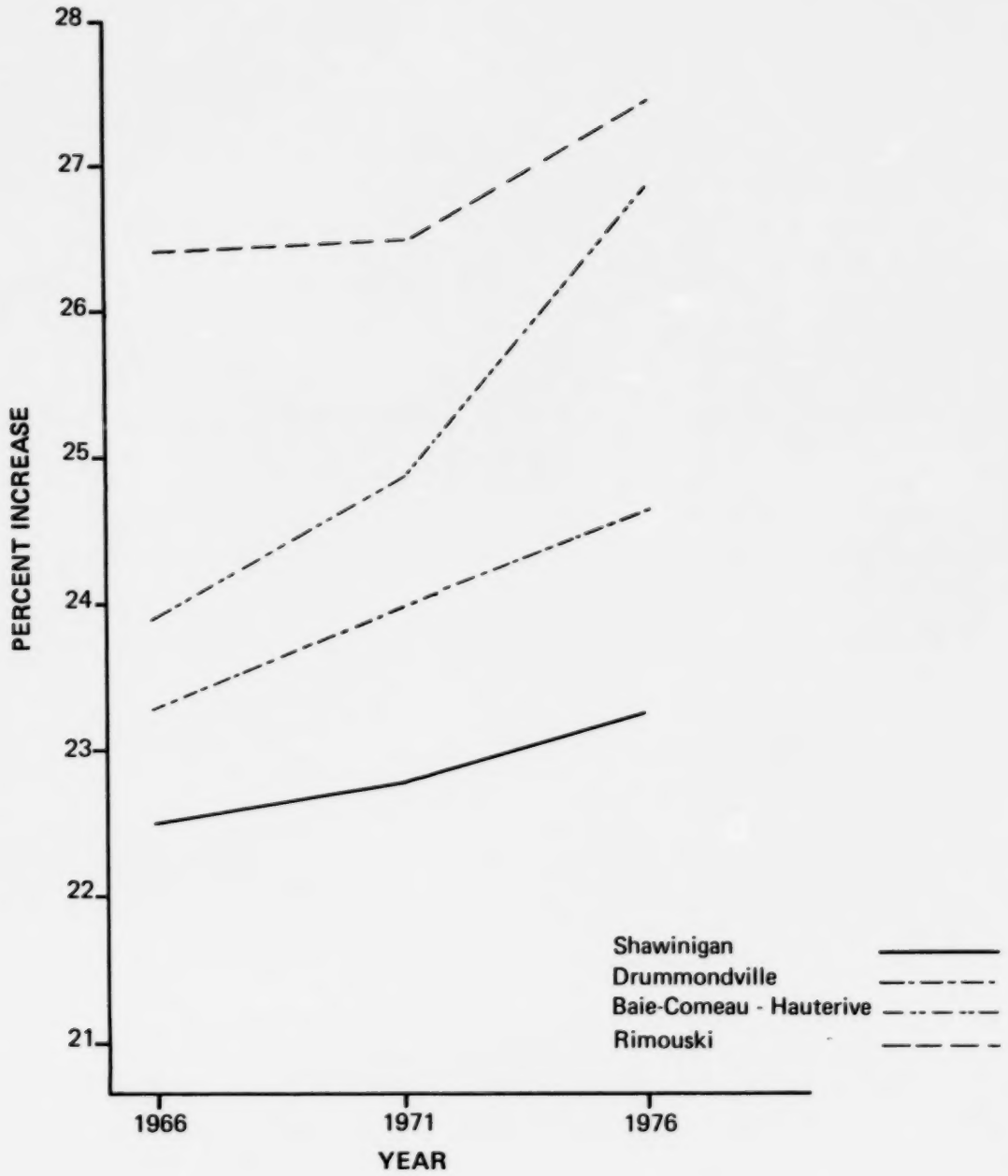


Figure A-1. Increment of the populations of females aged 15 to 44 years, from 1966 to 1976.

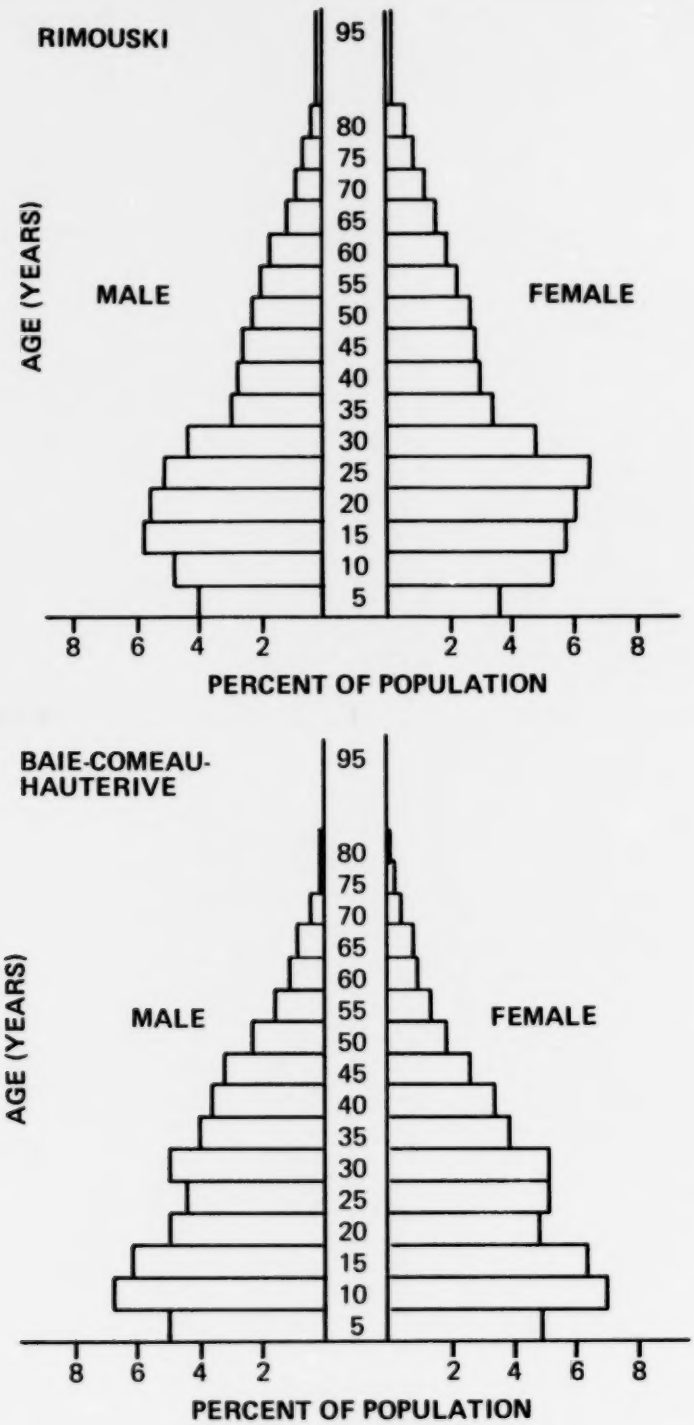


Figure A-2. Age and sex distributions of the populations of Rimouski and Baie-Comeau--Hauterive.

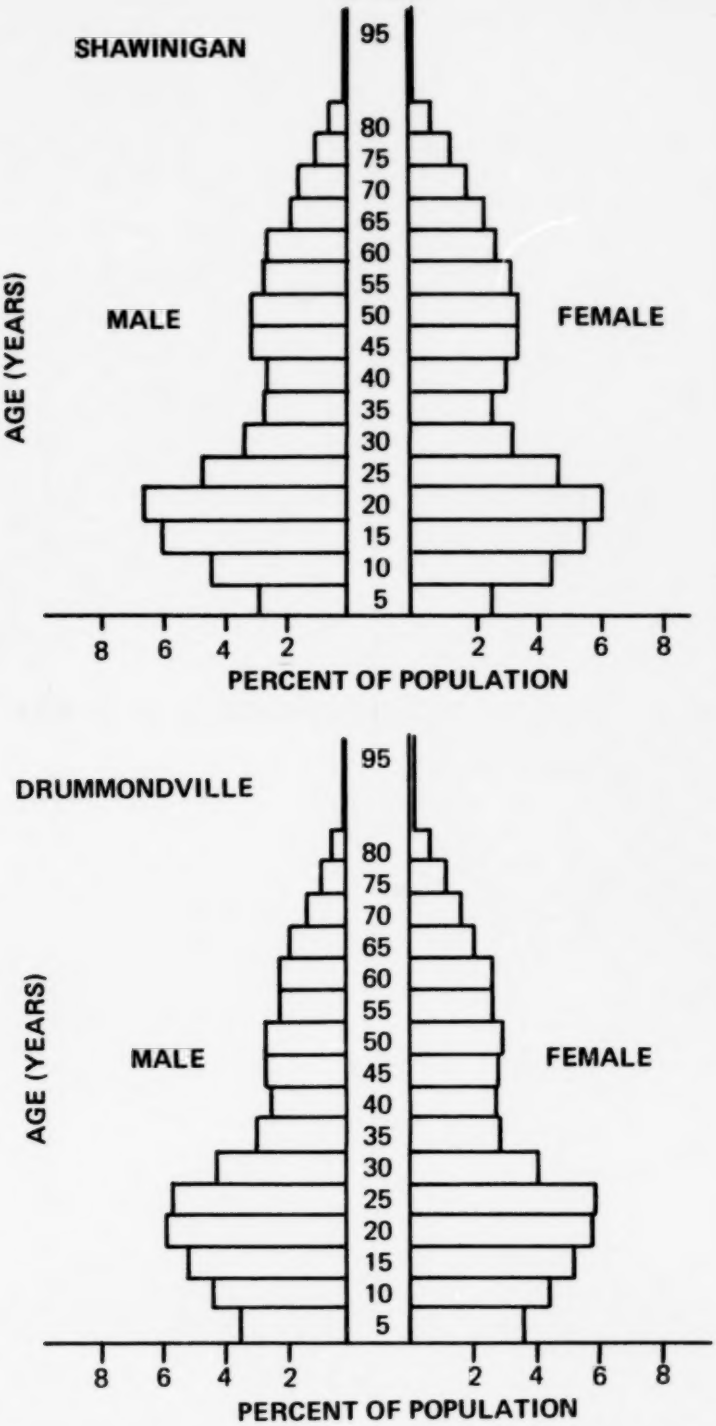


Figure A-3. Age and sex distributions of the populations of Shawinigan and Drummondville.

PART A. FORM TO BE FILLED BY CONSULTATION OF HOSPITAL RECORDS

I - TO BE FILLED FOR EVERY BIRTH DEFECT CASE

FILE NO. _____

FATHER'S NAME _____
Name Surname

MOTHER'S MAIDEN NAME _____
Name Surname

MOTHER'S ADDRESS (IF SHAWINIGAN RESIDENT) _____

_____

MOTHER'S AGE _____ years old

NEW-BORN: SEX M ☐ F ☐

HOSPITAL _____

FILE NO. _____

DATE OF BIRTH _____ _____ _____
Day Month Year

GESTATION AGE _____ weeks

VITAL STATUS AT BIRTH: Alive ☐ Dead ☐

BIRTH DEFECTS : DIAGNOSIS

1. _____
2. _____
3. _____
4. _____
5. _____

DELIVERY PHYSICIAN _____

PHYSICIAN IN CHARGE OF THE NEW-BORN AT THE HOSPITAL _____

_____

Figure A-4. Sample of questionnaire used in case-control study.

II - TO BE FILLED FOR BIRTHS AFTER JANUARY 1st 1975.

MOTHER'S CURRENT ADDRESS _____

_____

PHONE NUMBER : _____

MOTHER'S DATE OF BIRTH: _____ _____ _____
Day Month Year

FATHER'S DATE OF BIRTH: _____ _____ _____
Day Month Year

OTHER INFORMATIONS PERMITTING TO TRACE THE MOTHER:

_____

_____

OTHER RELATIVES' NAMES	ADDRESS	PHONE NO.
1. _____	_____	_____
2. _____	_____	_____
3. _____	_____	_____

HEALTH INSURANCE NO. _____

SOCIAL INSURANCE NO. _____

ETHNIC ORIGIN _____

RELIGION _____

ATTENDANT PHYSICIAN DURING PREGNANCY _____

_____

NO OF PREVIOUS DELIVERY _____

NO OF LIVING BIRTH _____

NO OF STILL BIRTH _____

NO OF SPONTANEOUS ABORTION _____

NO OF PREVIOUS CHILDREN WITH BIRTH DEFECTS _____

IF THERE ARE PREVIOUS BIRTH DEFECTS, DESCRIBE THE TYPE OF MAL-FORMATION

1. _____

2. _____

3. _____

Figure A-4. (continued).

ILLNESS AND TREATMENTS DURING PREGNANCY(*)

	DIAGNOSIS	TREATMENT
1.	_____	_____
2.	_____	_____
3.	_____	_____
4.	_____	_____

(*) PHYSICIANS COULD BE CONSULTED IF NECESSARY.

Figure A-4. (continued).

PART B — QUESTIONNAIRE TO BE FILLED DURING THE MOTHER'S INTERVIEW — FILE NO. _____

B - 1 RESIDENTIAL HISTORY OF THE MOTHER

B - 1.1 WHERE DID THE MOTHER RESIDE DURING EACH THIRD OF HER PREGNANCY?

	NO	STREET	CITY
FIRST THREE MONTHS			
SECOND THREE MONTHS			
THIRD THREE MONTHS			

B - 2 MEDICAL HISTORY OF THE MOTHER

B - 2.1 HAS MOTHER BEEN TREATED FOR (BEFORE OR DURING PREGNANCY)

- . tuberculosis ☐
- . epilepsy ☐
- . cancer ☐
- . mental illness ☐
- . does not remember ☐

IF APPLICABLE FILL THE FOLLOWING TABLE:

	Illness	Diagnosis Date	Treatment
1.			
2.			
3.			

B - 2.2 HAS MOTHER BEEN ILL DURING PREGNANCY? (ATTENTION TO VIRAL DISEASES, GERMAN MEASLES, MEASLES, MUMPS, HEPATITES)

YES ☐ NO ☐ DOESN'T REMEMBER ☐

Figure A-4 (continued).

IF YES, FILL THE FOLLOWING TABLE:

	Illness	Diagnosis Date	Treatment
1.			
2.			
3.			

TREATING PHYSICIAN DURING PREGNANCY _____

B - 2.3 DOES THE MOTHER REMEMBER SOMEONE AMONG HER RELATIVES WHO HAS AN ILLNESS OR A PHYSICAL DEFECT SINCE BIRTH:

YES ☐ DOES NOT KNOW ☐

NO ☐ REFUSE TO ANSWER ☐

IF YES, FILL THE FOLLOWING TABLE:

	Illness or Defect	Relationship
1.		
2.		
3.		

B - 3 MOTHER'S EXPOSURE TO RISK FACTORS

B - 3.1 HAS THE MOTHER EVER TAKEN L.S.D.?

YES ☐ NO ☐ REFUSE TO ANSWER ☐

IF YES:

. STARTING DATE _____

. STOPPING DATE (IF SHE STOPPED) _____

B - 3.2 HAS THE MOTHER BEEN X-RAYED DURING PREGNANCY?

YES ☐ NO ☐ DOES NOT REMEMBER ☐

Figure A-4. (continued).

IF YES, FILL THE FOLLOWING TABLE:

	Type of X-rays	Date
1.		
2.		
3.		

B - 3.3 SMOKING HABITS OF THE MOTHER

SMOKER ☐ EX-SMOKER ☐ NON-SMOKER ☐

IF SMOKER OR EX-SMOKER, HAS SHE SMOKED DURING THE 12 MONTHS PREVIOUS TO PREGNANCY?

YES ☐ NO ☐ DOES NOT REMEMBER ☐

IF YES, SHE SMOKED _____ CIGARETTES DAILY.

THERE WAS A CHANGE IN HER SMOKING HABITS DURING PREGNANCY

YES ☐ NO ☐ DOES NOT REMEMBER ☐

IF YES, FILL THE FOLLOWING TABLE:

	Week of pregnancy during which the change took place	Cigarettes daily after the change
Stops smoking		
Reduces smoking		
Increases smoking		
Begins smoking		

B - 3.4 MOTHER'S ALCOHOL INGESTION HABITS

HAS THE MOTHER USED ALCOHOLIC BEVERAGES REGULARLY (AT LEAST ONE A WEEK) DURING PREGNANCY?

YES ☐ NO ☐ REFUSE TO ANSWER ☐

IF YES, FILL THE FOLLOWING TABLE:

	Weekly amount
BEER	
WINE	
ALCOHOL	

Figure A-4. (continued).

B - 4 MOTHER'S WORK HISTORY

B - 4.1 HAS THE MOTHER WORKED OUTSIDE HOME DURING THE FIVE YEARS PRECEDING HER PREGNANCY?

YES ☐ NO ☐

IF YES, FILL THE FOLLOWING TABLE

	Employer	Work Done	Starting date	Ending date
1.				
2.				
3.				

B - 4.2 HAS THE MOTHER WORKED OUTSIDE HOME DURING PREGNANCY?

YES ☐ NO ☐

IF YES, FILL THE FOLLOWING TABLE:

	Employer	Work Done	Starting Date	Ending date
1.				
2.				
3.				
4.				

B - 5 FATHER'S DATA

B-5.1 FATHER'S DATE OF BIRTH _____ _____ _____
Day Month Year

B - 5.2 DOES THE FATHER KNOW SOMEONE OF HIS RELATIVES WHO HAS PRESENTED AN ILLNESS OR A PHYSICAL DEFECT SINCE BIRTH?

YES ☐ DOES NOT KNOW ☐

NO ☐ REFUSES TO ANSWER ☐

Figure A-4. (continued).

IF YES, FILL THE FOLLOWING TABLE:

	Illness or Physical defect	Relationship (with the father)
1.		
2.		
3.		

B - 5.3 FATHER'S WORKING HISTORY:

FILL THE FOLLOWING TABLE IN CHRONOLOGICAL ORDER WITHOUT LEAVING ANY GAP.

	Employer	Work done	Starting date	Ending date
1.				
2.				
3.				
4.				

Figure A-4. (continued).

APPENDIX B

DATA

CONTENTS

		PAGE
Table B-1.	Number of live births by sex, city, and year	B-3
Table B-2.	Number of stillbirths by sex, city, and year	B-4
Table B-3.	Number of live births in Shawinigan by month and year	B-5
Table B-4.	Number of live births in Drummondville by month and year	B-6
Table B-5.	Number of live births in Rimouski by month and year	B-6
Table B-6.	Number of births in Shawinigan by school district and year	B-7
Table B-7.	Distribution of children with central nervous system defects by sex and mother's residence at delivery	B-8
Table B-8.	Distribution of children with cardiovascular defects by sex and mother's residence at delivery	B-9
Table B-9.	Distribution of children with gastrointestinal defects by sex and mother's residence at delivery	B-10
Table B-10.	Distribution of children with urogenital defects by sex and mother's residence at delivery	B-11
Table B-11.	Distribution of children with defects of musculoskeleton by sex and mother's residence at delivery	B-12
Table B-12.	Distribution of children with defects of the mouth and upper airways by sex and mother's residence at delivery	B-13

(continued)

CONTENTS (continued)

PAGE

Table B-13. Distribution of children with defects of the eye or ear by sex and mother's residence at delivery B-14

Table B-14. Distribution of children with defects of the chest by sex and mother's residence at delivery B-14

Table B-15. Distribution of children with syndromes and other defects by sex and mother's residence at delivery B-15

Table B-16. Distribution of children with all types of birth defects B-15

TABLE B-1. NUMBER OF LIVE BIRTHS BY SEX, CITY, AND YEAR[a]

	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
Year	M	F	T	M	F	T	M	F	T	M	F	T
1966	235	214	449	307	296	603	374	341	715	178	188	366
1967	205	162	367	294	255	549	331	325	656	227	226	453
1968	184	163	347	304	264	568	274	295	569	242	198	440
1969	194	161	355	274	256	530	271	271	542	227	201	428
1970	175	143	318	263	259	522	255	247	502	232	184	416
1971	145	138	283	281	233	514	275	209	484	239	212	451
1972	128	144	272	246	241	487	243	223	466	229	195	424
1973	119	140	259	216	235	451	241	242	483	225	216	441
1974	134	133	267	279	259	538	260	244	504	227	239	466
1975	187	151	338	351	308	659	257	230	487	253	237	490
1976	157	168	325	356	362	718	237	215	452	231	223	454
1977	148	157	305	402	372	774	304	240	544	233	243	476
1978	166	163	329	427	380	807	307	240	547	218	227	445
1979[b]	157	163	320	395	371	766	282	232	514	227	231	458
Total	2334	2200	4534	4395	4091	8486	3911	3554	7465	3188	3020	6208

[a]From the Quebec Population Registry, Statistics Canada, and Community Health Centers.

[b]Estimated as the mean for the three previous years.

TABLE B-2. NUMBER OF STILLBIRTHS BY SEX, CITY, AND YEAR[a,b]

	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
Year	M	F	T	M	F	T	M	F	T	M	F	T
1966	1	2	3	6	0	6	7	3	10	2	2	4
1967	1	1	2	3	9	12	6	4	10	4	3	7
1968	1	0	1	3	2	5	2	0	2	6	3	9
1969	2	1	3	6	1	7	3	4	7	2	0	2
1970	1	0	1	0	6	6	3	1	4	0	0	0
1971			5			6	2	0	2			3
1972			2			12	3	2	5			3
1973			4			8	1	0	1			5
1974			2			9			6			8
1975			3			6			3			0
1976			2			2			4			3
1977			2			12			6			1
1978			1			2			5			2
1979[c]			2			5			5			2
Total			33			98			70			49

[a]Stillbirth is defined from 1971 to 1975 as > 20 weeks of gestation and from 1975 to 1978 as > 500 grams.

[b]Data sources: 1966 through 1970 (for Baie-Comeau--Hauterive, through 1973): Statistics Canada; 1971 through 1978 (for Baie-Comeau--Hauterive, 1974 through 1978): Quebec Population Registry.

[c]Estimated as the mean of the previous three years.

TABLE B-3. NUMBER OF LIVE BIRTHS IN SHAWINIGAN BY MONTH AND YEAR[a]

Month	1966	1967	1968	1969	1970	1971	1972	1973	1974	1975	1976	1977	1978	1979	Total
January	35	21	18	33	26	25	19	16	28	24	27	29	23	26	350
February	21	29	32	26	26	20	26	22	22	20	26	25	32	28	355
March	44	34	33	29	25	21	20	26	23	27	25	27	23	25	382
April	50	26	45	41	25	27	22	19	25	37	44	25	29	33	448
May	42	32	34	28	22	15	28	24	16	17	34	16	31	27	366
June	38	41	31	31	29	28	19	26	9	30	24	19	44	29	398
July	36	39	28	33	39	28	35	27	31	30	16	22	27	22	413
August	33	33	20	20	34	28	23	13	23	27	26	34	17	26	357
September	56	35	30	34	23	26	17	22	25	39	28	27	33	29	424
October	33	25	29	31	11	24	26	29	26	29	30	34	19	28	374
November	34	20	25	27	28	23	21	14	16	32	24	26	23	24	337
December	27	32	22	22	30	18	16	21	23	26	21	21	28	23	330
Total	449	367	347	355	318	283	272	259	267	338	325	305	329	320	4534

[a]From Quebec Population Registry.

TABLE B-4. NUMBER OF LIVE BIRTHS IN DRUMMONDVILLE BY MONTH AND YEAR[a]

Month	1971	1972	1973	1974	1975	1976	1977	Total
January	47	38	39	42	42	72	54	334
February	43	38	33	45	52	48	68	327
March	49	54	45	47	49	62	70	376
April	54	40	29	63	50	78	83	397
May	46	32	41	55	36	64	58	332
June	44	36	31	41	76	54	70	352
July	34	36	39	39	65	67	59	339
August	52	49	32	51	58	76	62	380
September	41	36	31	45	66	61	60	340
October	34	40	38	37	65	88	76	378
November	27	37	49	35	56	65	56	325
December	39	47	33	40	36	49	61	305
Total	510	483	440	540	651	784	777	4185

[a]From Quebec Population Registry.

TABLE B-5. NUMBER OF LIVE BIRTHS IN RIMOUSKI BY MONTH AND YEAR[a]

Month	1971	1972	1973	1974	1975	1976	1977	Total
January	39	31	33	31	36	51	32	253
February	35	29	33	39	29	40	47	252
March	37	33	48	34	40	44	47	283
April	38	32	36	39	49	39	48	281
May	34	38	44	57	45	41	46	305
June	34	33	29	43	41	38	39	257
July	38	34	38	38	42	39	33	262
August	39	23	41	38	37	44	42	264
September	48	44	34	44	48	44	41	303
October	37	45	40	30	44	28	50	274
November	35	35	30	37	37	36	32	242
December	32	39	23	37	42	36	28	237
Total	446	416	429	467	490	480	485	3213

[a]From Quebec Population Registry.

TABLE B-6. NUMBER OF BIRTHS IN SHAWINIGAN BY SCHOOL DISTRICT AND YEAR

Year	School District															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Unknown	Total
1966	21	18	15	35	49	53	49	42	17	48	22	10	23	25	25	452
1967	22	15	15	21	25	41	39	42	18	28	14	9	34	17	29	369
1968	13	18	16	13	24	37	47	31	42	36	8	11	34	12	6	348
1969	29	5	13	23	28	50	40	45	14	27	16	5	24	11	28	358
1970	14	10	17	19	24	32	32	37	10	23	14	9	18	15	45	319
1971	20	2	8	15	20	42	28	26	17	34	11	6	25	8	26	288
1972	25	6	10	17	26	34	25	25	12	21	12	11	19	18	13	274
1973	20	10	10	14	17	26	31	23	12	24	8	21	26	17	4	263
1974	12	6	11	18	25	36	20	21	23	14	14	13	27	19	10	269
1975[a]	15	10	13	20	30	37	29	37	19	20	14	22	30	23	22	341
1976	15	13	12	16	28	29	30	43	10	21	10	26	25	21	28	327
1977[a]	16	10	10	15	23	25	32	34	12	21	11	29	25	24	19	307
1978	18	9	8	16	20	23	38	28	15	23	14	34	29	29	26	330
1979[a]	17	9	9	16	22	24	35	31	14	22	13	32	28	27	23	322
Total	257	141	167	258	361	489	475	465	235	362	181	238	368	266	304	4567
Total plus proportional number of unknown	275	151	179	276	387	524	509	498	252	388	194	255	394	285	--	4567

[a]Estimated numbers.

TABLE B-7. DISTRIBUTION OF CHILDREN WITH CENTRAL NERVOUS SYSTEM DEFECTS BY SEX AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
Type of Birth Defect	M	F	T	M	F	T	M	F	T	M	F	T
Anencephalia	2	4	6	5	5	10	1	4	5	1	2	3
Encephalocele (meningocele, myelomeningocele)	6	5	11	4	14	18	8	9	17	4	4	8
Spina bifida	0	1	1	1	2	3	3	1	4	1	0	1
Hydrocephalus	7	9	16	3	12	15	5	3	8	5	3	8
Microcephalia	0	0	0	0	0	0	0	0	0	1	3	4
Other central nervous system defects (macrocephalia, aplasia of pituitary gland, cyclopia, unspecified severe cerebral defect)	1	0	1	1	0	1	0	0	0	1	0	1
Total	13	17	30	12	25	37	16	14	30	10	11	21

TABLE B-8. DISTRIBUTION OF CHILDREN WITH CARDIOVASCULAR DEFECTS BY SEX AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
Type of Birth Defect	M	F	T	M	F	T	M	F	T	M	F	T
Hyper- and hypoplasia of the heart	0	1	1	1	1	2	1	1	2	1	0	1
Atrioventricular canal	2	1	3	0	0	0	0	0	0	0	1	1
Patent ductus arteriosus	1	1	2	1	0	1	1	1	2	2	1	3
Cardiac valve anomalies (atresia of the tricuspid valve)	0	1	1	1	0	1	1	0	1	0	0	0
Transposition of the large vessels	2	2	4	2	1	3	0	0	0	0	0	0
Coarctation of the aorta	1	2	3	3	0	3	0	0	0	0	1	1
Pulmonary stenosis	1	1	2	0	0	0	1	1	2	1	2	3
Aortic stenosis or atresia	0	0	0	1	1	2	0	0	0	0	0	0
Cor bioloculare or triloculare	2	2	4	0	0	0	1	0	1	0	1	1
Ventricular and/or atrial septal defect (CIV-CIA)	10	5	15	12	10	22	5	2	7	10	7	17
Murmurs of the heart	2	2	4	0	0	0	1	5	6	14	14	28
Unspecified cardiovascular defects	1	0	1	2	2	4	2	1	3	2	2	4
Other cardiovascular system malformations (defect of the aorta, Fallot's tetralogy, ectopia cordis, fibroelastosis of the heart, dextraposition of the aorta)	0	1	1	3	0	3	1	1	2	1	1	2
Total	21	16	37	22	15	37	11	12	23	30	29	59

TABLE B-9. DISTRIBUTION OF CHILDREN WITH GASTROINTESTINAL DEFECTS BY SEX AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

Type of Birth Defect	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
	M	F	T	M	F	T	M	F	T	M	F	T
Tracheo-esophagus fistula	0	0	0	0	1	1	1	0	1	1	0	1
Esophagus atresia	0	0	0	1	1	2	0	0	0	0	1	1
Pyloric stenosis	0	0	0	1	0	1	1	1	2	0	1	1
Duodenum atresia	0	0	0	0	1	1	2	0	2	0	0	0
Large intestine atresia	1	0	1	0	1	1	0	0	0	0	0	0
Congenital diaphragmatic hernia	2	3	5	4	1	5	2	0	2	0	2	2
Congenital megacolon	1	0	1	0	1	1	0	1	1	0	0	0
Meckel's diverticulum	1	0	1	0	0	0	0	0	0	1	0	1
Congenital inguinal hernia	1	0	1	0	0	0	1	0	1	2	0	2
Congenital umbilical hernia	0	0	0	3	2	5	1	2	3	0	4	4
Defects of the anus	2	1	3	0	1	1	1	1	2	3	1	4
Liver and biliary duct defects	1	0	1	0	0	0	1	0	1	1	0	1
Congenital diastasis rectiabdominis	0	0	0	0	1	1	0	1	1	0	0	0
Total	7	4	11	8	9	17	10	6	16	7	8	15

TABLE B-10. DISTRIBUTION OF CHILDREN WITH UROGENITAL DEFECTS BY SEX AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

Type of Birth Defect	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
	M	F	T	M	F	T	M	F	T	M	F	T
Hypospadias	23	-	23	24	-	24	19	-	19	8	-	8
Epispadias	0	-	0	1	-	1	0	-	0	0	-	0
Other defects of the penis	0	-	0	2	-	2	1	-	1	0	-	0
Clitorism	-	0	0	-	1	1	-	0	0	-	0	0
Internal defects of the genitalia (enlargement of the uterus or the vagina)	-	1	1	-	0	0	-	0	0	-	0	0
Imperforate hymen	-	1	1	-	0	0	-	0	0	-	1	1
Hermaphrodism	1	0	1	1	0	1	1	0	1	1	1	2
Defects of the kidney, ureter, urinary bladder, and urethra (example: hydronephrosis agenesia)	3	1	4	4	3	7	1	0	1	6	1	7
Polycystic kidney	2	2	4	1	1	2	0	1	1	0	1	1
Implantation of the colon into the urinary bladder	0	0	0	1	0	1	0	0	0	0	0	0
Total	28	2	30	31	5	36	22	1	23	14	4	18

TABLE B-11. DISTRIBUTION OF CHILDREN WITH DEFECTS OF MUSCULOSKELETON BY SEX AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
Type of Birth Defect	M	F	T	M	F	T	M	F	T	M	F	T
Bone defect of the skull (absence of skull, craniosynostosis, cranial asymmetry)	1	1	2	1	0	1	0	3	3	1	3	4
Syndactyly	3	1	4	6	5	11	2	0	2	3	1	4
Polydactyly	1	4	5	3	1	4	3	7	10	3	4	7
Adactyly partial or complete	0	1	1	1	1	2	1	0	1	2	1	3
Clubfoot	10	10	20	18	15	33	7	7	14	14	5	19
Other defects in the positioning of the foot and lower limbs	2	3	5	4	7	11	3	8	11	3	5	8
Defects of the ribs and clavicle	0	1	1	0	0	0	1	0	1	0	0	0
Congenital dislocation of the hip and related defects of the hip	0	7	7	1	5	6	0	2	2	1	9	10
Congenital torticollis	1	1	2	0	0	0	0	2	2	2	0	2
Phocomelia	0	0	0	1	0	1	1	2	3	0	1	1
Other defects of the musculoskeletal system (hypoplasia of one arm, anteversion of the collum femoris, congenital knee luxation, facial asymmetry, deformities of the vertebrae, arthrogryposis, agenesia or defects of the hand, scoliosis, deformity of the thorax)	3	5	8	0	1	1	2	3	5	2	0	2
Total	18	31	49	35	34	69	16	30	46	27	28	55

TABLE B-12. DISTRIBUTION OF CHILDREN WITH DEFECTS OF THE MOUTH AND UPPER AIRWAYS BY SEX AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

Type of Birth Defect	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
	M	F	T	M	F	T	M	F	T	M	F	T
Cleft palate, cleft lip	3	6	9	4	3	7	10	3	13	5	6	11
Micrognathia	1	0	1	0	0	0	0	0	0	0	1	1
Stenosis of the nasal cavities	0	1	1	0	0	0	0	1	1	0	0	0
Absence of soft palate	0	1	1	0	0	0	0	0	0	0	0	0
Congenital laryngeal stridor	2	3	5	4	2	6	2	2	4	1	3	4
Other defects of the mouth and the upper airways (choanal atresia, hypertrophy of the conchae, deviation of the nasal septum)	0	0	0	0	0	0	2	2	4	0	3	3
Total	6	11	17	8	5	13	14	8	22	6	12	18

TABLE B-13. DISTRIBUTION OF CHILDREN WITH DEFECTS OF THE EYE OR EAR BY SEX AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
Type of Birth Defect	M	F	T	M	F	T	M	F	T	M	F	T
Anophthalmus	1	0	1	0	0	0	0	0	0	0	0	0
Bischler's cataract	3	1	4	0	0	0	0	0	0	0	0	0
Defect of the external auditory canal	0	1	1	1	0	1	3	0	3	0	0	0
Dacryostenosis	0	0	0	1	0	1	0	0	0	0	0	0
Total	4	2	6	2	0	2	3	0	3	0	0	0

TABLE B-14. DISTRIBUTION OF CHILDREN WITH DEFECTS OF THE CHEST BY SEX AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
Type of Birth Defect	M	F	T	M	F	T	M	F	T	M	F	T
Pneumomediastinum	1	0	1	0	0	0	0	0	0	0	0	0
Absence of the lung	0	0	0	1	0	1	0	0	0	0	1	1
Other defects of the chest (absence of lobe in the lung, honeycomb lung)	0	2	2	0	0	0	0	0	0	0	0	0
Total	1	2	3	1	0	1	0	0	0	0	1	1

TABLE B-15. DISTRIBUTION OF CHILDREN WITH SYNDROMES AND OTHER DEFECTS BY SEX AND MOTHER'S RESIDENCE AT DELIVERY (1966-1979)

	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
Type of Birth Defect	M	F	T	M	F	T	M	F	T	M	F	T
Down's syndrome	1	1	2	6	4	10	4	4	8	2	4	6
Trisomia and other chromosomal aberrations	0	1	1	0	0	0	3	1	4	1	4	5
Hyperplasia, hypoplasia, and absence of adrenal gland	0	1	1	0	1	1	0	0	0	1	1	2
Congenital goiter	0	0	0	0	1	1	0	0	0	0	0	0
Other unclassified birth defects (congenital lymphedema, thymic enlargement, generalized hypoplasia, melanoma, situs inversus, atresia of papilla mammae, multiple birth defects)	0	2	2	0	1	1	3	2	5	1	1	2
Total	1	5	6	6	7	13	10	6	16	5	10	15

TABLE B-16. DISTRIBUTION OF CHILDREN WITH ALL TYPES OF BIRTH DEFECTS (1966-1979)

	Mother's Residence at Delivery											
	Shawinigan			Drummondville			Baie-Comeau--Hauterive			Rimouski		
Type of Birth Defect	M	F	T	M	F	T	M	F	T	M	F	T
Total number of children with birth defects	85	74	159	109	84	193	92	71	163	87	83	170

APPENDIX C

SUMMARY OF A STUDY OF THE FEASIBILITY OF MEASURING AMBIENT VINYL CHLORIDE CONCENTRATIONS IN SHAWINIGAN

This study was conducted by Gilles P. Thériault, Department of Social and Preventive Medicine, and Jean-Guy Guillot and Réjean Harvey, Laboratory of Toxicology, Laval University, Quebec, Canada, under Contract No. 68-02-2780 for the U.S. Environmental Protection Agency Health Effects Research Laboratory, Research Triangle Park, North Carolina (Project Officer, Gregg Wilkinson).

INTRODUCTION

The purpose of this study was to determine whether the concentration of vinyl chloride monomer (VCM) in the ambient air of Shawinigan, Quebec, is sufficient to be detected through currently available field sampling techniques. In the first phase of the study, an analytical method was established for measuring very weak concentrations of VCM in the laboratory. Standard curves were obtained for VCM in air and on activated charcoal, and results were verified by an independent laboratory. The second phase was to develop a field sampling method appropriate to the levels of VCM in Shawinigan and to the sensitivity of the analytical methods. Finally, ambient VCM concentration was studied with respect to wind velocity and atmospheric inversion, and samples taken simultaneously indoors and outdoors were compared.

Field sampling was done with Bendix BDX 44 Super Sampler pumps, VCM was adsorbed on activated charcoal, and samples were analyzed with a Perkin-Elmer model F-42 headspace analyzer gas chromatograph. The desorbent was N,N-dimethylacetamide in water.

CONCLUSIONS AND RECOMMENDATIONS

The concentration of VCM in ambient air in Shawinigan can be measured. Levels of 5 to 45 ppb were detected in this study. VCM on activated charcoal was detected in quantities as small as 62.4 ng desorbed in a 24-ml vial. For air sampling, the optimum flow rate was 100 cc/min, using varying sampling times. Results can be affected by sampling time and conditions (temperature and relative humidity), which must thus be closely monitored. Rapid freezing of samples minimizes VCM migration on activated charcoal.

The dispersion of VCM and its concentration in ambient air appeared to be strongly influenced by wind, atmospheric inversion, and local topography.

High wind velocity results in rapid dilution of the pollutant, while atmospheric inversion limits vertical mixing. Local topographic features make conventional dispersion models inadequate for estimating VCM levels. Simultaneous indoor and outdoor sampling at a Shawinigan residence revealed lower VCM concentrations indoors than outdoors (this observation should be confirmed under more favorable sampling conditions).

VCM was found in snow sampled near the VCM plant, suggesting that 1) when snow melts, VCM could become concentrated in the runoff, thus contaminating local drinking water, and 2) liquefied VCM in snow near the plant could be released into the atmosphere when the temperature rises, raising the atmospheric VCM concentration.

A program of field sampling for VCM in Shawinigan should last at least one year, with 24-hour sampling at about ten topographically and demographically representative sites. VCM adsorbed on purified activated charcoal should be analyzed through a modified gas chromatography method using an appropriate precolumn and a capillary column. Sampling tubes should be transported and stored at -20°C or below, and samples should be analyzed as soon after sampling as possible. Ideally, a meteorological station should be installed near the VCM source. In addition, a program to measure VCM in snow and water around the plant and in the plant's wastewater would be desirable.

APPENDIX D

DISPERSION PATTERNS OF VINYL CHLORIDE EMITTED BY B.F. GOODRICH CO., LTD., SHAWINIGAN, QUEBEC

by

Donald Labrie and Bernard Coupal
Envirobec, Inc.

CONTENTS

	PAGE
Introduction	D-2
Description of the Mathematical Model	D-2
Meteorology	D-7
Plant Parameters	D-13
Topography	D-13
Results and Discussion	D-17
References	D-19

INTRODUCTION

As part of an epidemiological study carried out by a medical team from Laval University, we were asked to calculate dispersion of vinyl chloride emitted by the B.F. Goodrich Co., Ltd., polyvinyl chloride plant in Shawinigan, Quebec.

We first estimated vinyl chloride emission over the past twelve years and then estimated population exposure using a dispersion model. The calculations depend on many assumptions and on partial meteorological data. No emissions data or production records were available from B.F. Goodrich. Because information on such parameters as emission heights, gas velocity and temperature, and building heights (for evaluating plume height and the effects of buildings on dispersion) were not available to us, we used an emission factor estimated from survey data (1), and the plume height and effects of buildings were not evaluated.

Meteorological data were also partial. Historical series on wind direction and speed were available for one full year. Records of cloud cover and sunshine hours were not available for calculating the stability classes, so the conditions prevailing at Quebec Airport were used instead. These results should be used only for comparing various areas surrounding the plant.

To simulate the dispersion patterns, we have used a climatic model; this means that the concentration profiles generated by the mathematical model are valid for a long exposure. They do not simulate dispersion patterns that might exist under special conditions such as inversions. Thus, they should not be compared with experimental determinations for short sampling times and for specific meteorological conditions prevailing for a given period.

DESCRIPTION OF THE MATHEMATICAL MODEL

Equation and Basic Assumption

The mathematical model used in the present study is an integrated form of the continuity equation applied to a continuous emission source. It is based on the Gaussian assumption that dispersion patterns follow normal distributions, both vertically and in the direction perpendicular to the wind motion. The model, called the Pasquill-Gifford equation, is as follows:

$$C(x,y,z,H) = \frac{Q}{2\pi\sigma_y\sigma_z u} \exp\left[-1/2\left(\frac{y-y_s}{\sigma_y}\right)^2\right] \left\{\exp\left[-1/2\left(\frac{z-z_s-H}{\sigma_z}\right)^2\right] + \exp\left[-1/2\left(\frac{z-z_s+H}{\sigma_z}\right)^2\right]\right\} \quad (1)$$

where C = concentration, grams per cubic meter,
x,y,z = position of the receptor,
H = effective height of the stack (physical height plus the thermal plume), meters,
Q = continuous source strength, grams per second,
u = wind speed, meters per second,
σ_y,σ_z = dispersion coefficients, meters, and
x_s,y_s,z_s = position of the stack, meters.

The x-axis is oriented in the wind direction, while the z-axis is vertical. The y-axis is normal to the x-axis.

The basic assumptions for Equation 1 are as follows:

1. Normal distribution for dispersion in the y and z directions.

2. The height of the thermal plume is calculated separately from Equation 1, using the thermal and kinetic content of the plume. The height is then added to the physical height of the stack.

3. The flow rate of the source is uniform.

4. The pollutants are chemically inert.

5. The wind speed is constant.

6. The pollutants are reflected by the ground surface.

7. The topography is flat.

Dispersion Coefficients and Sampling Time

Dispersion coefficients are the controlling parameters of Equation 1, and their numerical evaluation is related to the stability of air (2). Ideally, the dispersion coefficients should be determined experimentally by monitoring variation in the wind direction (3). However, in the present study, they are estimated from known correlations. Two sources are available: the power law and the Pasquill formula.

Power law. These equations are recommended by the American Society of Mechanical Engineers (3). The equations have the following form:

$$\sigma = ax^b \qquad (2)$$

where a and b are related to climatic conditions, and x is the distance from the receptor to the emission source. The climatic conditions are described by the classes of stability. Table D-1 lists the values for a and b for each of four stability classes. The subscripts y and z refer to the horizontal and vertical dispersion coefficients.

TABLE D-1. DISPERSION COEFFICIENTS RELATED TO STABILITY CLASSES

Dispersion Coefficient	A-B Strongly Unstable	B-C Weakly Unstable	D Neutral	E-F Stable
a_y	0.40	0.36	0.32	0.31
b_y	0.91	0.86	0.78	0.71
a_z	0.40	0.33	0.22	0.06
b_z	0.91	0.86	0.78	0.71

In the equations, the distance x and the value of σ are given in meters. They are valid for distances up to 10 km for high sources. The dispersion coefficients calculated with Equation 2 and data from Table D-1 are valid for sampling times of one hour.

Pasquill formulas. The formulas proposed by Pasquill are as follows:

$$\sigma_y = \exp (a_y - b_y \ln(x)) \qquad (3a)$$

$$\sigma_z = \exp (a_z - b_z \ln(x) - C_z \ln^2(x)) \qquad (3b)$$

where exp is an exponential, ln is the natural logarithm, x is the distance in kilometers, and the dispersion coefficients are given in meters. These equations are valid for distances up to 10 km and sampling times of 1 h.

Table D-2 gives the values for a and b by stability class.

TABLE D-2. DISPERSION COEFFICIENTS (PASQUILL) RELATED TO STABILITY CLASSES

Dispersion Coefficient	A Strongly Unstable	B Moderately Unstable	C Slightly Unstable	D Neutral	E Stable	F Very Stable
a_y	5.340	5.090	4.636	4.214	3.912	3.509
b_y	0.865	0.875	0.899	0.901	0.904	0.905
a_z	6.053	4.707	4.117	3.411	3.058	2.618
b_z	2.009	1.059	0.920	0.732	0.679	0.658
c_z	0.223	0.011	-0.002	-0.032	-0.045	-0.054

Interpolation for different classes of stability. The equations (especially the power law) do not encompass all the stability classes, necessitating interpolation. The program for the model uses either form of equation to calculate the dispersion coefficients.

Sampling time. For sampling times longer than 1 h, the dispersion coefficients must be modified. The dispersion coefficient in the vertical direction is used without any change, while the model is integrated in the direction normal to the wind direction. In so doing, the model assumes that the dispersion distributes the pollutants uniformly in that direction. The integration is usually carried out within a wind sector of 22.5°. This procedure is standard for converting the model for short sampling times into an expression for longer exposure (4).

Plume Height

Upon release from the stack, the pollutants have an exit velocity and a certain heat content, which generates a vertical motion of the pollutants. The height is calculated using various equations that take into account both the thermal and kinetic content of the plume. Since no information on the heat and kinetic content of the plume was available for the present study, the calculation of the thermal plume was not made. Buildings surrounding the emission sources may influence the effective height of the source. This factor was not considered in the present study, because of lack of information.

Wind Intensity

The wind intensity used in the calculations was measured at a height of 10 m and corrected to the physical height of the stack. In other words,

$$u \text{ average} = u_1 \, (H/Z_1)^c \qquad (4)$$

where u average = mean wind velocity at height H,
H = physical height of the source, meters,
u_1 = wind velocity at 10 m height (Z_1), and
c = a parameter related to class stability (0.5 for stable conditions [Class F] and 0.25 for unstable conditions [Class B]). The value of the parameter is adjusted for various stability classes using the following relationships:

Pasquill Class:	A	A-B	B	B-C	C	D	E	F
Numerical equivalent (P):	1	2	3	4	5	7	9	11

For each stability class, the parameter C is given by

$$C = \frac{(P + 3)}{24} \qquad (5)$$

For example, for Class C (P = 5), we have

$$C = 8/24 = 0.33 \qquad (6)$$

Long-Term Concentration

Long-term concentrations are obtained for any direction by multiplying the wind frequency in that direction by the concentration obtained after integration in a wind sector, as explained in the paragraph "Sampling Time," above.

Weather Inversions

The dispersion of pollutants in atmospheric air takes place in a given volume whose magnitude is related to stability class. The mixing depth of the dispersion space is an important parameter that must be supplied to the model. It is assumed that the pollutants, while dispersing, are reflected both on the ground and on the lid of the mixing space.

Influence of Topography

The Pasquill-Gifford equation assumes a flat terrain. To take into account the influence of the topography, the equation of motion would have to be integrated with the equation of continuity. This approach is complex, and there are seldom data on wind turbulence. It is, however, possible to use experimental corrections such as those suggested by the National Oceanic and Atmospheric Administration of the United States (NOAA) (2). These corrections are incorporated in the model; additional comments will be made when the topography of the site is discussed.

Stability Classes

Stability classes are used in the model to calculate the dispersion coefficients and to correct the wind velocity. Table D-3 gives the stability classes for various conditions of wind velocity and percentage of sunshine.

The average concentration for a long sampling time must take into account changes in stability class during the period considered. This is done by summarizing the various concentrations according to the following equation:

$$C \text{ average} = \sum_{L=1}^{5} C(L) * \text{Per}(L) \qquad (7)$$

where C average = long-term concentration,
C(L) = long-term concentration for stability class L, and
Per (L) = frequency of class L.

TABLE D-3. STABILITY CLASSES

Windspeed (mph)	Sunshine			Night
	Strong	Moderate	Weak	
< 4.5	A	A-B	B	F
4.5 - 6.7	A-B	B	C	E
6.7 - 11.2	B	B-C	C	D
11.2 - 13.4	C	C-D	D	D
> 13.4	D	D	D	D

METEOROLOGY

Wind data (speed and direction) were taken in Shawinigan by the Sérvices de Protection de l'Environnement du Québec (SPEQ) from November 1977 to October 1978. Measurements were made hourly at a station (No. 823) approximately 1 km N-NW of the site, at an altitude of 100 m. The plant itself is at an altitude of 93 m. As described in the section on the mathematical model, the estimation of stability classes required information on wind cover and sunshine. As this information was not available for Shawinigan or Trois-Rivières, information from the airport in Quebec City was used instead.

Figures D-1 through D-5 are the wind roses obtained from Station No. 823. Figure D-1 is the annual wind rose, and Figures D-2 through D-5 are the seasonal wind roses. These figures show rather uniform wind patterns, with four dominant wind directions (SW, NW, N, and NE). The wind speed was fairly constant at 3 m/s.

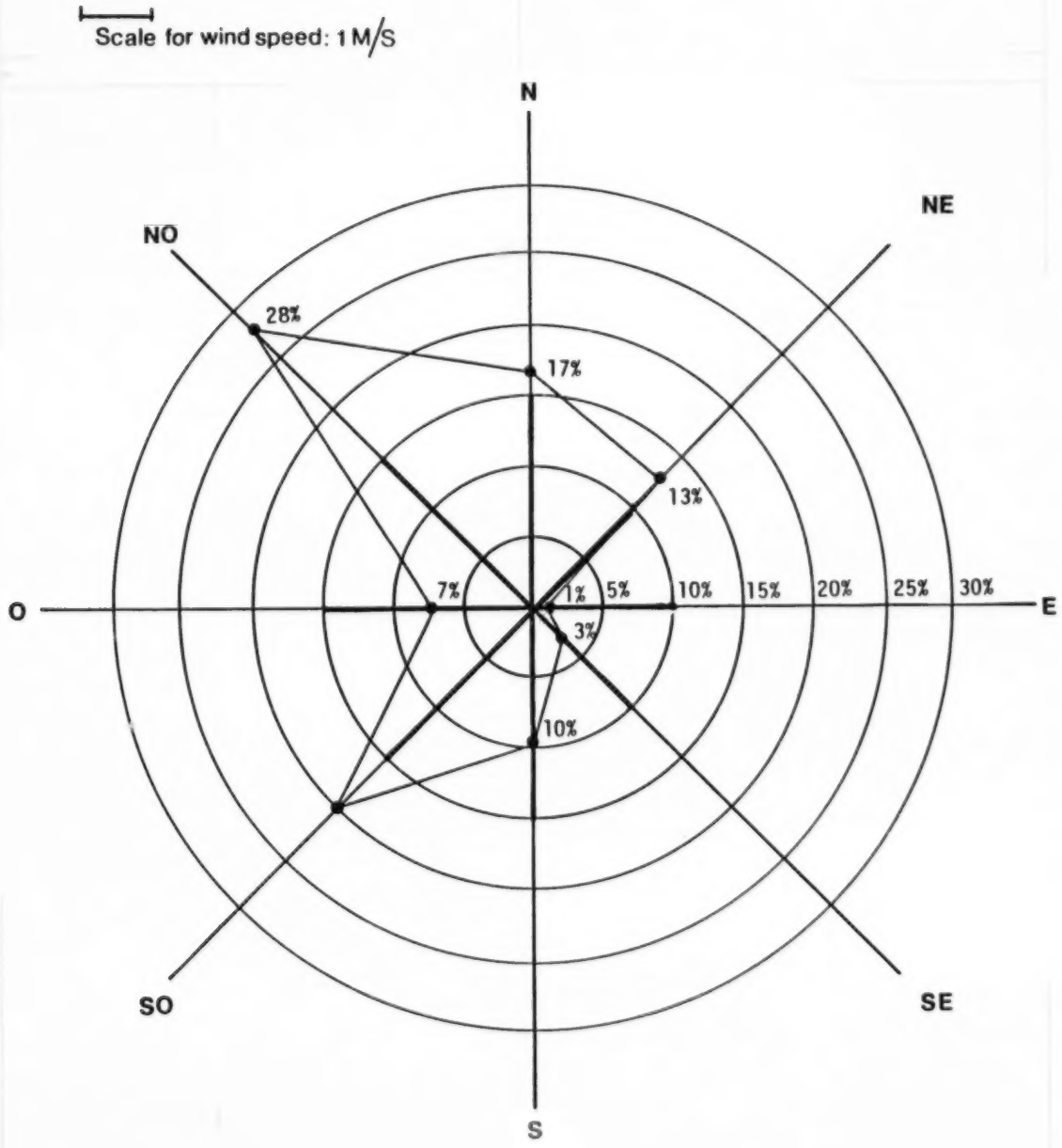


Figure D-1. Annual wind rose for Shawinigan, Station No. 823.

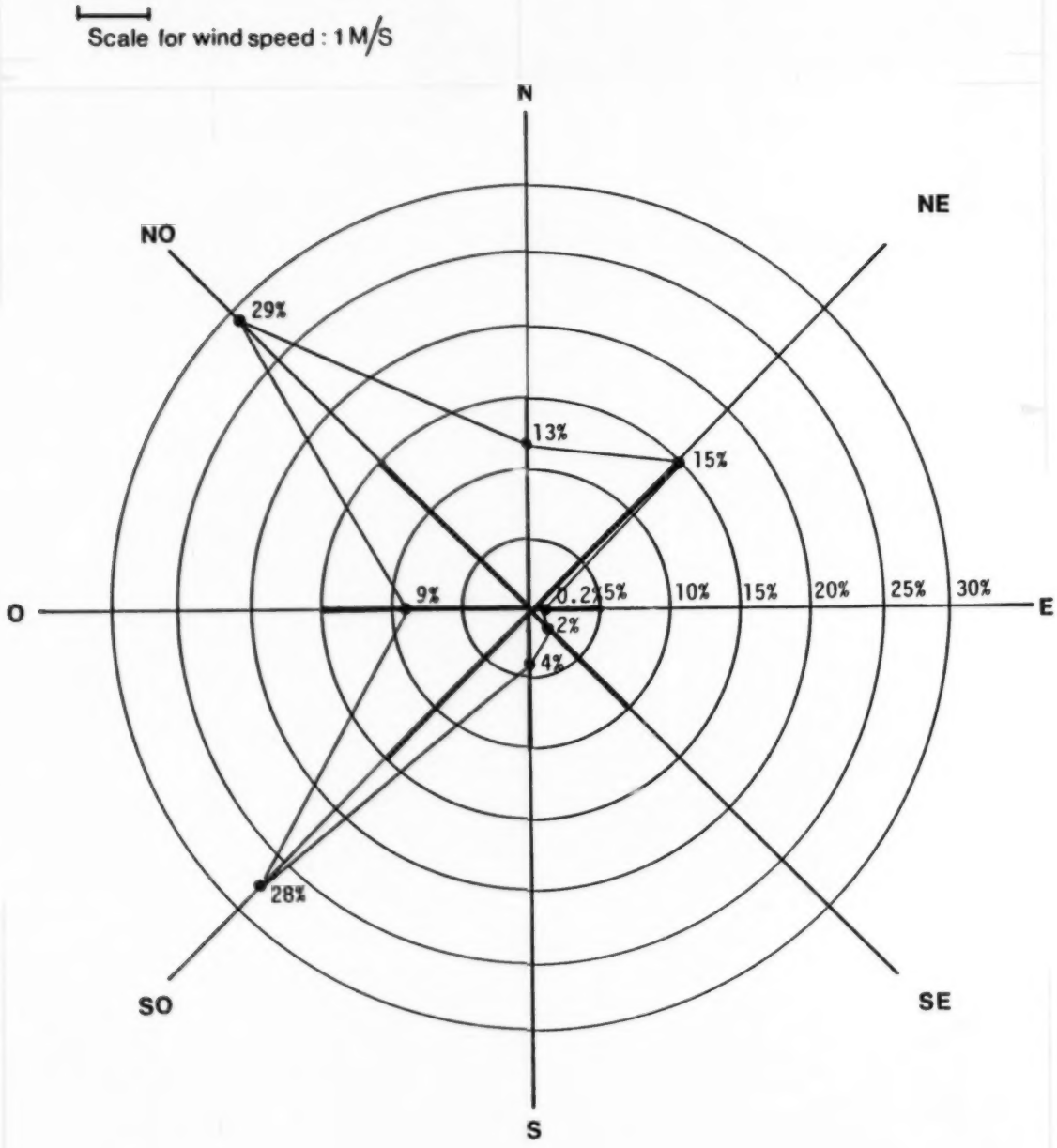


Figure D-2. Winter wind rose for Shawinigan, Station No. 823.

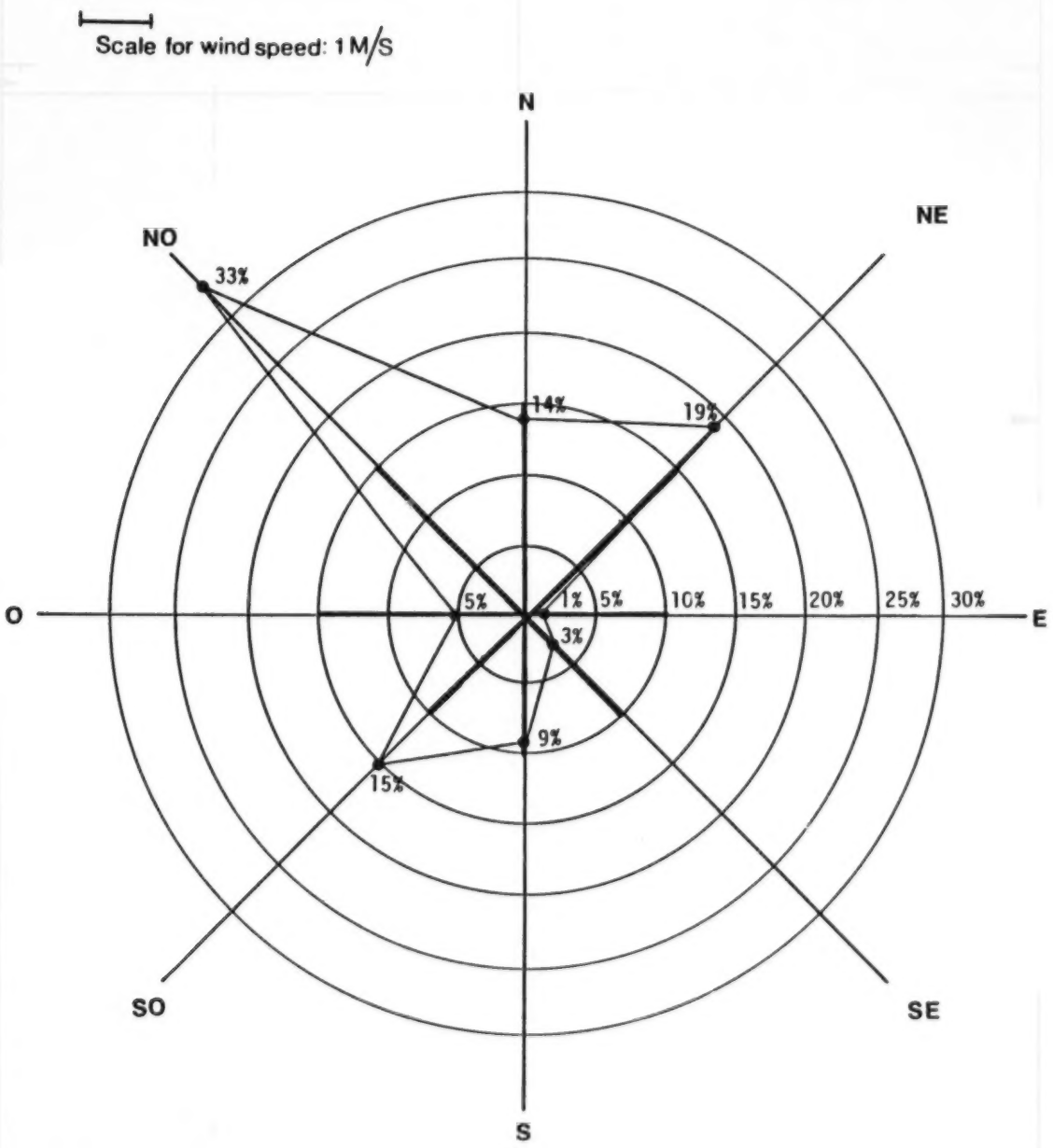


Figure D-3. Spring wind rose for Shawinigan, Station No. 823.

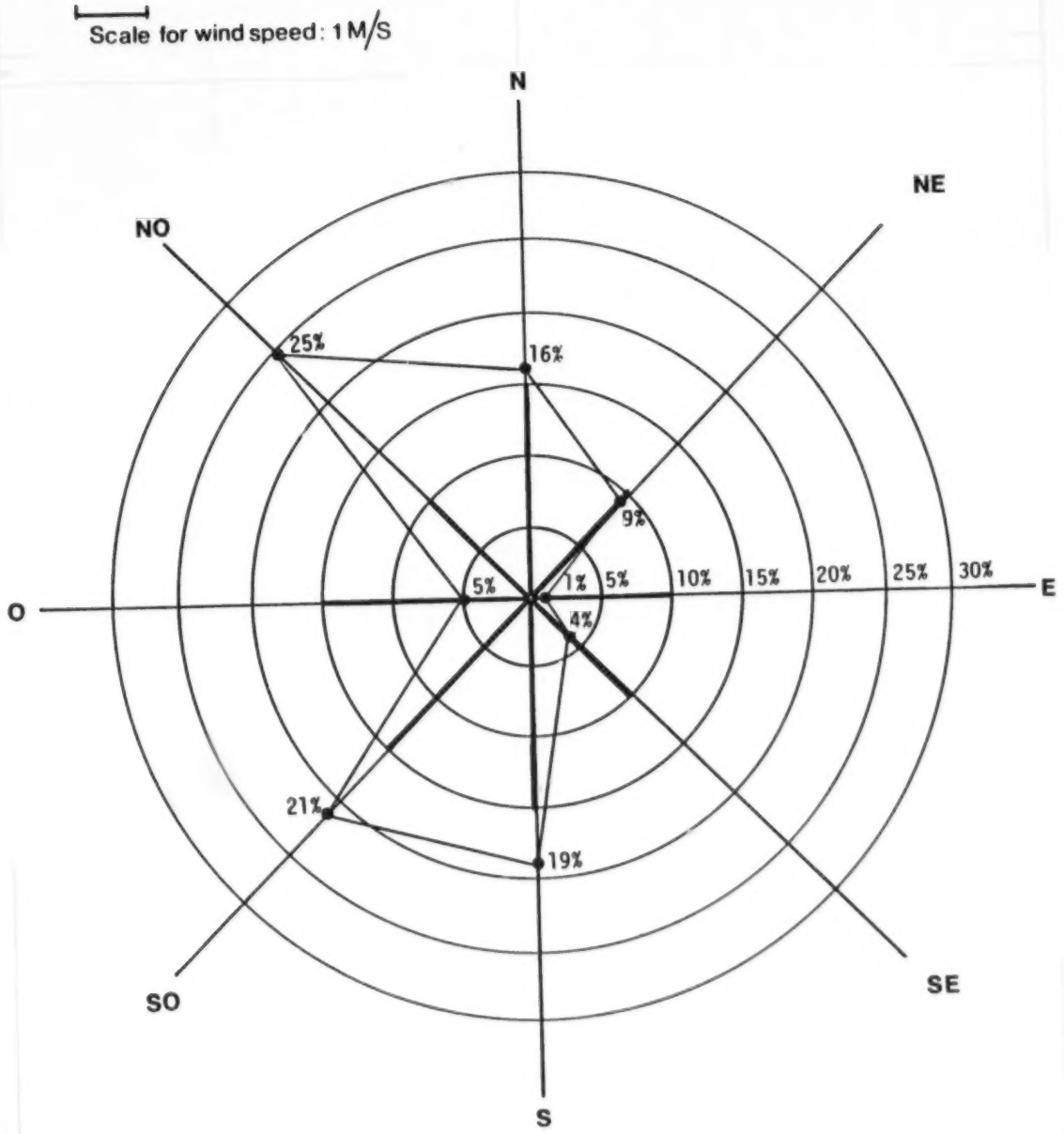


Figure D-4. Summer wind rose for Shawinigan, Station No. 823.

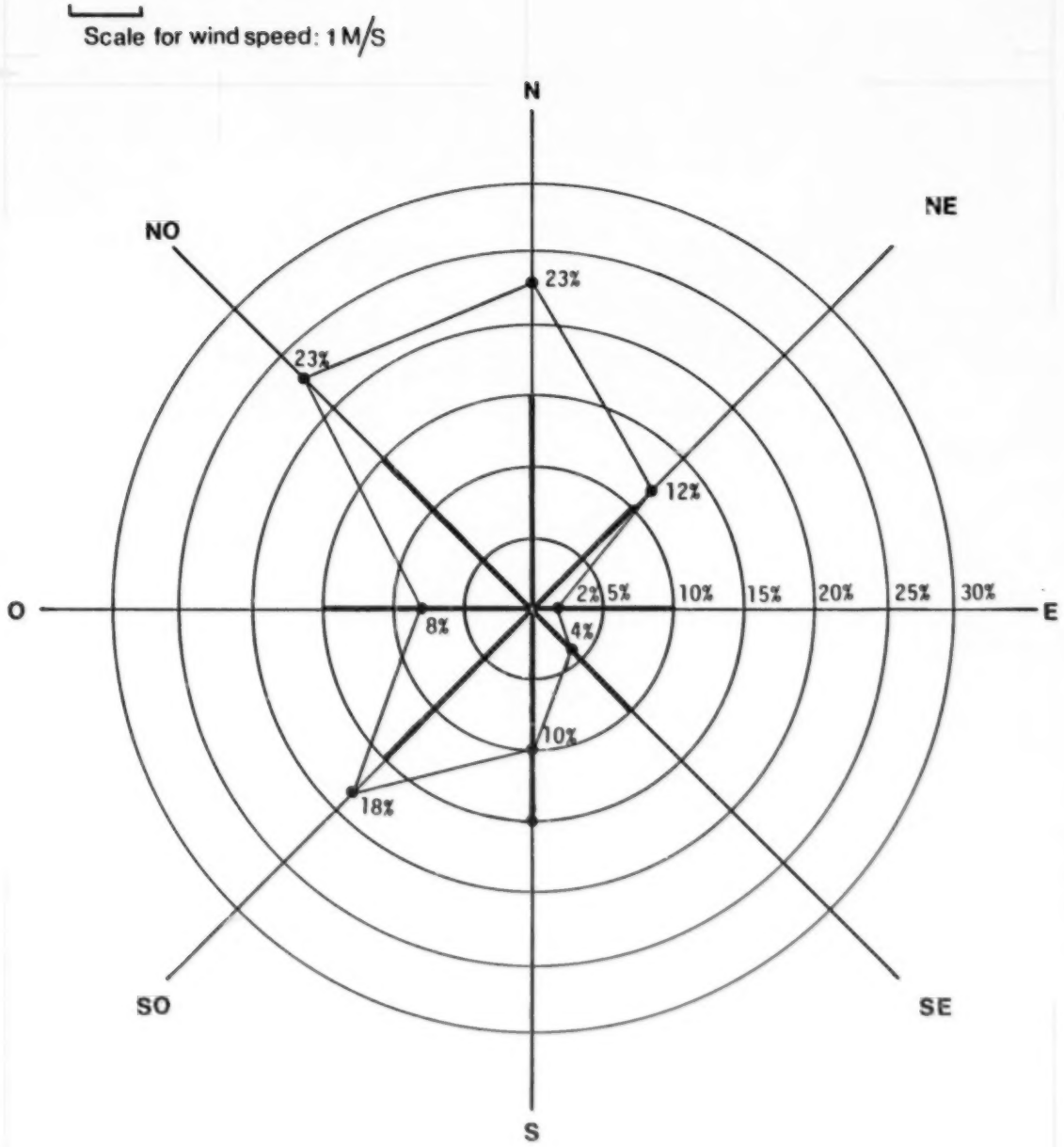


Figure D-5. Fall wind rose for Shawinigan, Station No. 823.

PLANT PARAMETERS

As the rate of emission of pollutants from the plant was not available, it was estimated from data published by Environment Canada (1). An overall emission factor of 3.9 lb/100 lb of polyvinyl chloride produced was used. In response to the EPS Petrochemical/Synthetic Resin Industries Atmospheric Emission and State-of-the-Art Questionnaire in 1974, three out of four Canadian polyvinyl chloride producers reported estimates of their vinyl chloride emissions for 1973. The weighted average rate of captive emissions and emergency venting resulting from plant malfunctions were computed to be 2.34 lb/100 lb of polyvinyl chloride produced. Very little information pertaining to fugitive emissions was given. These data are very comparable to those for American plants, as reported by the U.S. Environmental Protection Agency. Based on the American survey, EPS estimated vinyl chloride losses from Canadian plants through fugitive emissions to be 1.53 lb/100 lb of polyvinyl chloride produced.

Because production records from the B.F. Goodrich plant were not available, the annual PVC production was assumed to be constant since 1966 and was estimated at 50,000,000 lb. According to the Environment Canada report (1), this figure represents the production for 1973. Calculated on a continuous basis, the emission rate is

$$\frac{3.9}{100} \times \frac{50 \times 10^6}{\text{year}} \times \frac{454 \text{ grams}}{\text{lbs.}} \times \frac{10^6 \text{ µg}}{\text{gram}} \times \frac{\text{year}}{365 \text{ days}} \times \frac{\text{day}}{24 \text{ h}} \times \frac{\text{h}}{3{,}600 \text{ s}}$$

$$= 28 \times 10^6 \text{ µg/s}$$

Because no information was available on the thermal and kinetic content of the plume, it was impossible to estimate the plume height, which was thus neglected. The effective height of the stack was taken as the physical height, estimated at 45 m (150 ft) (5).

TOPOGRAPHY

The site is at an altitude of 93 m. The topography of the surroundings is highly variable, and the elevation profiles of the dominant wind directions are shown in Figures D-6, D-7, and D-8. In the NW and N directions, there are areas where the elevation is identical to that of the plant elevation plus the stack height. The pollutants are thus dispersing at a very low height above these areas; this must be considered in the calculations.

As discussed above, the Pasquill-Gifford equation was originally designed for flat topography. To take into account changes in elevation, a procedure was used similar to the approach used by NOAA (2). The stack height is reduced to correct for the elevation, as illustrated in Figure D-9.

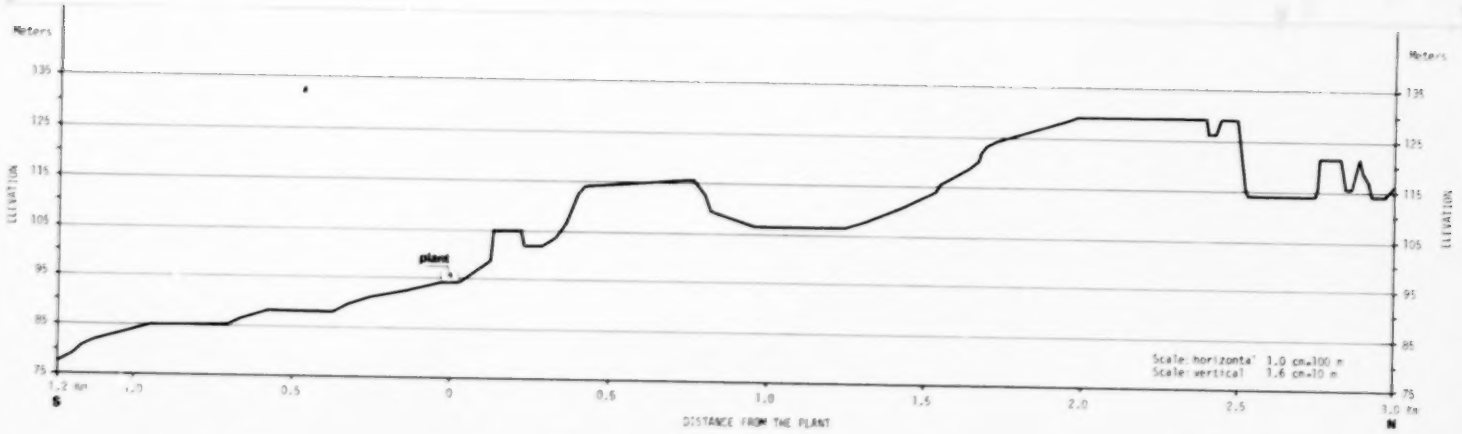


Figure D-6. Elevation profile: south-north.

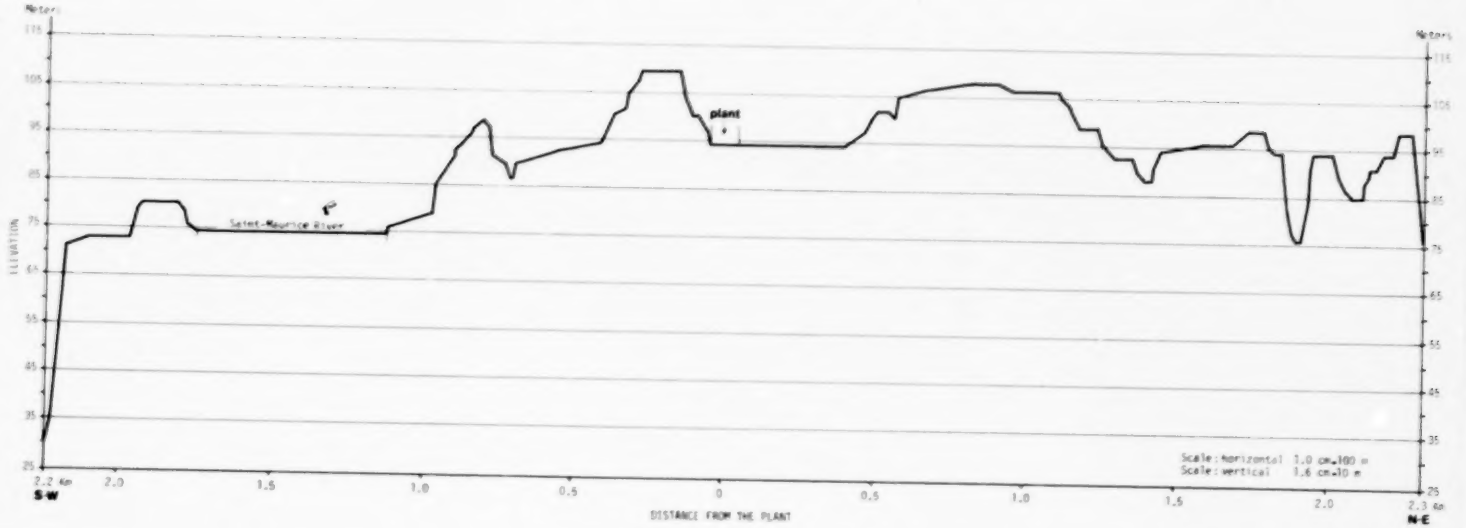


Figure D-7. Elevation profile: southwest-northeast.

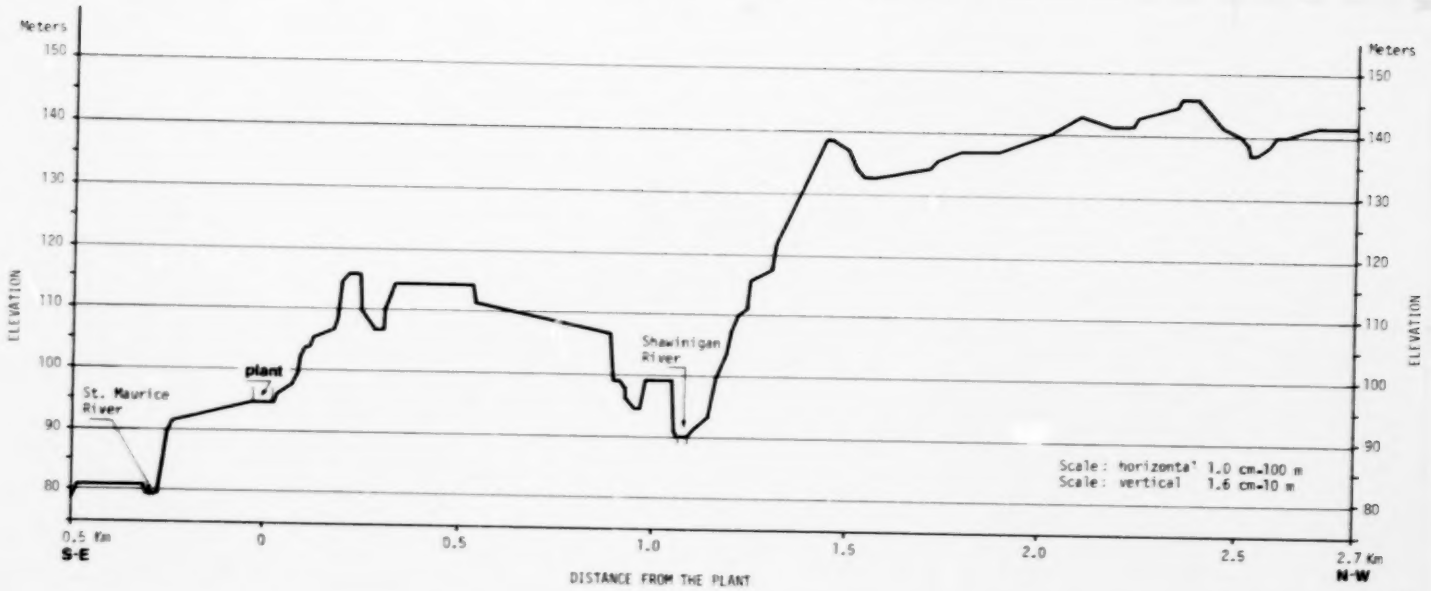
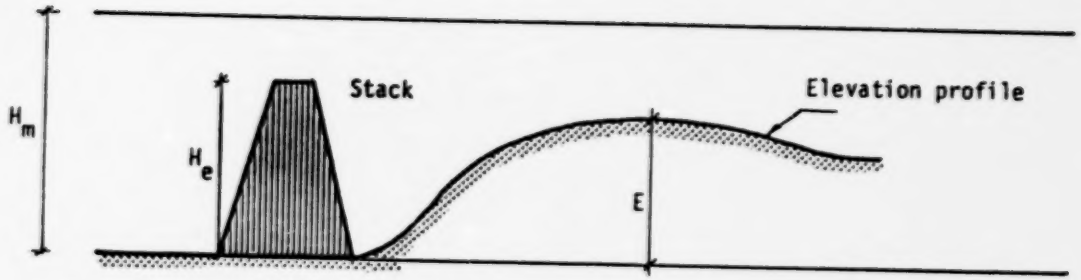


Figure D-8. Elevation profile: southeast-northwest.

REAL CASE:

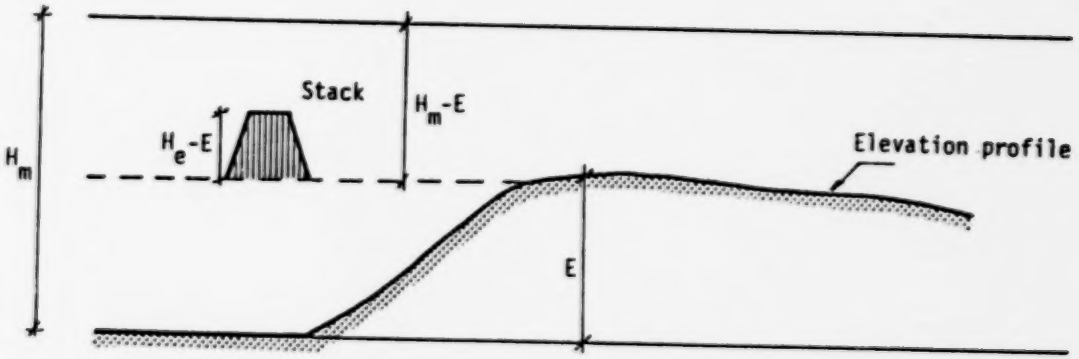
Inversion lid



Reference: elevation of plant site

IDEALIZED CASE:

Inversion lid



Reference: elevation of plant site

Figure D-9. Method of correction for topography.

RESULTS AND DISCUSSION

Dispersion patterns were simulated for various distances from the plant, using mixing heights of 500 m during the day and 250 m at night. Dispersion coefficients calculated with both ASME and Pasquill formulas were used in the model, and the higher value of concentration retained. Annual and seasonal wind roses were used with the model; as the wind roses are all fairly similar, (see Figures D-1 through D-5), the simulations did not generate significantly different dispersion patterns (within 10%). Figure D-10 shows the yearly average concentration profiles for vinyl chloride. Examination of this figure reveals the following:

1. The pollutants move mainly over the sectors defined by the S, E, and N directions, because the wind blows mainly from the NW, SW, N, and W.

2. The highest concentrations are found to the SE and S, because the wind is most frequently from the NW and N. The topography in these directions, however, favors dispersion, because the mixing height increases. If the topography were flat, the concentration field would be higher.

3. The wind blowing from the NE is significant, and averages 14%. Nonetheless, the concentration profile in the SW direction is low, probably because a cliff SW of the plant stops the pollutant.

In Table D-4, the school districts, shown on Figure D-10, are compared with respect to average concentration of vinyl chloride. Districts 3 and 5 should be subdivided, because they are located in an area where there are important variations in concentration. District 5, for instance, is roughly divided in half by the 2-ppb isopleth. District 3 lies under the highest concentration profiles generated by the simulation. Subdivision would show areas with concentrations as high as 60 ppb.

It should be kept in mind that the concentration profiles shown in Figure D-10 were obtained using a yearly average wind rose and should not be compared with experimental determinations derived for specific sets of meteorological conditions. Moreover, the lack of data on plant parameters has been a major drawback in the simulation; real data would be necessary for more accurate calculations. The same caution applies to meteorological conditions. All the calculations have been carried out using data for a short period; additional data from Station No. 823 and from other sites (to assess more completely the influence of the topography) would be needed.

Figure D-10. Isopleths of vinyl chloride.

TABLE D-4. YEARLY AVERAGE CONCENTRATION OF VINYL CHLORIDE BY SCHOOL DISTRICT

School District	Yearly Average Concentration of Vinyl Chloride (ppb)	
	Minimum	Maximum
1	5	15
2	-	(< 2)
3	2	≈ 60
4	-	(< 2)
5	1.5	4
6	1.5	3
7	3	4
8	2	3
9	-	(< 2)
10	-	(< 2)
11	-	(< 2)
12	-	(< 2)
13	-	(< 2)
14	-	(< 2)
15	-	(< 2)

REFERENCES

1. Environment Canada. Air Pollution Emissions and Control Technology: Vinyl Chloride Industry, Economic and Technical Review. Report EPS-3-AP-77-4, March 1978.

2. Slade. Meteorology and Atomic Energy. U.S. Atomic Energy Commission, 1968.

3. ASME. Recommended Guide for the Prediction of the Dispersion of Airborne Effluents. Maynard Smith, 1968.

4. Turner. Workbook of Atmospheric Dispersion Estimates. U.S. Department of Health, Education, and Welfare, 1969.

5. Thériault, G., R. Harvey, and J.G. Guillot. Exposition de la population de Shawinigan au chlorure de vinyle. Université Laval, 1979.